МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
УО «ВИТЕБСКИЙ ГОСУДАРСТВЕННЫЙ ОРДЕНА ДРУЖБЫ НАРОДОВ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

Профессиональные болезни: тесты с разъяснениями

Occupational diseases: explanatory tests

Рекомендовано учебно-методическим объединением по высшему медицинскому, фармацевтическому образованию в качестве пособия для студентов учреждений высшего образования, обучающихся по специальности 1-79 01 01 «Лечебное дело»

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В пособии представлены контрольные тесты и даны разъяснениями по общим вопросам профессиональной патологии, диагностике, лечению и профилактике профессиональных заболеваний, возникающих от воздействия промышленных аэрозолей, вибрации, шума, функционального перенапряжения, химических веществ.

Пособие предназначено для студентов медицинских вузов, магистрантов, аспирантов, клинических ординаторов, врачей-интернов, а также врачей-терапевтов, врачей общей практики, -невропатологов, -оториноларингологов, -ортопедов и врачей других специальностей, участвующих в проведении медицинских осмотров, работе врачебно-консультационных и медико-реабилитационных экспертных комиссий.

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Control tests to section I

1. What formulation most fully answers the question - what are occupational diseases?
1. An isolated category of acute and chronic diseases resulting from exposure to the body of adverse factors of the working environment.
2. Any acute and chronic diseases that occur in persons working in conditions of exposure to the body of adverse factors of the working environment.
3. Chronic diseases of the internal organs, formed as a result of prolonged exposure of the body to working adverse factors of the working environment.
4. Diseases resulting from violation of safety regulations at work.
5. Acute and chronic diseases of internal organs that necessarily arise in professionally trained workers with long work experience in production.

2. In what century were the first publications made describing the clinical picture of occupational diseases?
1.14th century
2.15 th century.
3.16th century
4.17 th century
5.18th century

3. Who is the author of the first publications describing the clinical picture of occupational diseases in miners?
1. Paracelsus.
2.Agricola.
3.Vezaliy.
4.Pirogov.
5. Botkin.

4. Who is involved in the diagnosis of chronic occupational diseases?
1. District doctor.
2. Medical-consulting commissions (MCC) of general medical institutions.
3. Regional Centers of Occupational Pathology.
4. The Republican Center for Occupational Pathology and Allergy.
5. The Republican Center for Hygiene, Epidemiology and Public Health.

5. Who has the right to recognize in certain cases a professional disease as those diseases that are not included in the official list of occupational diseases?
1. District doctor.
2. MCC treatment-and-prophylactic institutions of general profile.
3. Regional Centers of Occupational Pathology.
4. The Republican Center for Occupational Pathology and Allergy.
5. Scientific Research Institute of Occupational Pathology.

6. Who supervises the establishment of a causal connection of diseases with the profession and compliance with instructions for applying the list of occupational diseases?

1. MCC treatment-and-prophylactic institutions of general profile.
2. Regional Centers of Occupational Pathology.
3. Territorial health authorities.
4. The Republican Center for Occupational Pathology and Allergy.
5. Scientific Research Institute of Occupational Pathology.

7. What primarily determines the risk of occupational disease?

1. From hygienic conditions in the workplace.
2. From the tension and severity of the labor process.
3. From the experience of work in adverse production conditions.
4. From the number of employees in an industrial enterprise.
5. From gender and age working in production.

8. What class correspond to safe working conditions, characterized by such production factors, which preserve the health of workers, and create prerequisites for maintaining a high level of efficiency?

1. 1st class.
2. 2nd class
3. 3rd class.
4. 4th class.
5. None of the above.

9. What class correspond to safe working conditions, characterized by such production factors, the levels of which do not go beyond the limits of hygienic standards, and possible changes in the functional state of the body, arising under their influence, are restored during regulated breaks or the beginning of the next shift and do not have an adverse effect in the near and distant period on the health status of workers and their offspring?

1. 1st class.
2. 2nd class
3. 3rd class.
4. 4th class.
5. None of the above.

10. What class do the working conditions correspond to when production factors at the workplace exceed hygienic standards and adversely affect the worker's body and / or its offspring?

1. 1st class.
2. 2nd class
3. 3rd class.
4. 4th class.
5. None of the above.

11. To what class do hazardous working conditions correspond, characterized by such production factors, the levels of which significantly exceed the limits of hygienic standards and the impact of which during the work shift (or its part) can pose a threat

to the life of an employee, the high risk of developing acute professional diseases, including severe forms?
1. 1st class.
2. 2nd class
3. 3rd class.
4. 4th class.
5. None of the above.

12. To what class are the working conditions characterized by such production factors, the levels of which have deviations from hygienic standards and the impact of which causes functional changes in the body, recovering, as a rule, with a longer one (shifts) interrupting contact with harmful factors and increase the risk of damage to health?
Class 1.3 - 1st degree.
Grade 2.3 - 2nd degree.
Grade 3.3 - 3rd degree.
Grade 4.3 - 4th degree.
5.4th grade.

13. To what class are the working conditions characterized by such production factors, the levels of which have deviations from hygienic standards and cause persistent functional changes in the body, leading in most cases to an increase in production-related morbidity, the appearance of initial signs or mild forms of occupational diseases that occur after prolonged exposure (often after 15 years or more)?
Class 1.3 - 1st degree.
Grade 2.3 - 2nd degree.
Grade 3.3 - 3rd degree.
Grade 4.3 - 4th degree.
5.4th grade.

14. To what class are the working conditions characterized by such production factors, the levels of which have deviations from hygienic standards and lead to the development of, as a rule, occupational diseases of mild and moderate degrees of severity (with loss of occupational ability) in the period employment, growth of chronic (work-related) diseases, including increased incidence rates with temporary disability?
Class 1.3 - 1st degree.
Grade 2.3 - 2nd degree.
Grade 3.3 - 3rd degree.
Grade 4.3 - 4th degree.
5.4th grade.

15. Which class includes working conditions characterized by such production factors, the levels of which have deviations from hygienic standards and under which severe forms of occupational diseases can occur (with loss of general working capacity), there is a significant increase in the number of chronic diseases and high levels morbidity with temporary disability?
Class 1.3 - 1st degree.
Grade 2.3 - 2nd degree.
Grade 3.3 - 3rd degree.
4. 3rd grade - 4th degree.
5.4th grade.

16. What diseases belong to the competence of the occupational physician?

1. Diseases caused by the action of a certain harmful factor of the working environment, having a fairly clearly defined, specific clinical picture.

2. Diseases resulting from exposure to harmful factors of the working environment, but in the clinical picture, which lack specific manifestations that unambiguously indicate the professional nature of this pathology.

3. Diseases that are not etiologically associated with adverse factors of production conditions, but that can occur in a more severe and severe form when exposed to such factors.

4. All listed.

5.None of the above.

17. What diseases are classified as acute occupational?

1. Occurring as a result of a single exposure to a damaging factor of high intensity.

2. As a result of the influence of a damaging factor during one work shift.

3. Affected by the impact of a damaging factor for a maximum of three work shifts.

4. Occurred with prolonged exposure to damaging factors of low intensity.

5. Occurred as a result of systematic exposure to the damaging factor of low intensity, which has cumulative properties.

6.All listed.

18. What diseases are classified as chronic occupational?

1. Occurring as a result of a single exposure to a damaging factor of high intensity.

2. As a result of the influence of a damaging factor during one work shift.

3. Occurred with long-term exposure to damaging factors of low intensity.

4. Occurred as a result of systematic exposure to the damaging factor of low intensity, which has cumulative properties.

5. All listed.

19. Which of the following is included in the list of harmful substances and adverse production factors capable of causing occupational diseases?

1. Chemical: benzene and its derivatives, mercury and its compounds, lead and its inorganic compounds, lead tetraethyl and others.

2. Biological: allergens for diagnosis and treatment, protein-vitamin concentrates, infected biological material, etc.

3. Industrial aerosols: silicon-containing, silicate and silicate-containing dust, organic and mineral dust, carbon black, dust of metals and their alloys, welding aerosol, etc.

4. Physical effects: sources of ionizing radiation, industrial vibration, industrial noise, etc.

5. Adverse factors of the labor process: physical overload, work associated with local muscular tensions, etc.

20. What are the features of the emergence and clinical course of occupational diseases in the modern conditions of high-tech manufacturing?

1. The risk of severe forms of occupational diseases has increased.

2. The time for the formation (experience) of severe occupational diseases and disabilities has decreased.

3. Mild forms of occupational diseases are more common.
4. Professional diseases are more likely to occur in young people who have recently begun work in adverse working conditions.
5. For patients with occupational diseases, prolonged work experience in unfavorable production conditions is characteristic.

21. What documents do not need the patient when he is sent to medical institutions that have the right to establish the diagnosis of chronic occupational disease?
1. Open sick leave.
2. The direction of the medical institution.
3. An extract from the ambulatory card drawn up by the attending physician.
4. Sanitary-hygienic characteristics of working conditions, compiled by the hygienist doctor of the center of hygiene and epidemiology.
5. An extract from the work record of the patient, confirming his work experience at the enterprise where a professional illness could have occurred.

22. What should be indicated in an extract from an ambulatory card prepared for sending a patient for examination to the Republican Center for Occupational Pathology and Allergology?
1. All diseases and the time of their occurrence;
2. Information about vaccinations and vaccinations.
3. Information on the state of health according to the results of the preliminary (upon entering employment) and subsequent medical examinations;
4. The clinical picture of the current disease, the results of special instrumental and laboratory studies;
5. The list of therapeutic measures and their effectiveness.

23. What should be done when establishing the case of a chronic occupational disease, first established?
1. Make and send a notice of chronic occupational disease to the insured.
2. Compile and forward emergency notification to the territorial center of hygiene and epidemiology.
3. To investigate the causes of the disease in the workplace.
4. Draw up a statement on the results of the investigation into the causes of occupational disease.
5. To provide the investigation materials to the Prosecutor's Office and the Republican Center for Occupational Pathology and Allergology.

24. Who investigates every case of acute or chronic occupational disease that is first established?
1.Party therapist.
2. Hygienist doctor of the center of hygiene and epidemiology.
3. State Labor Inspector.
4. Representatives of the Regional Center for Occupational Pathology or the Republican Center for Occupational Pathology and Allergology.
5. Representatives of the employer and the workforce.

25. To whom are copies of the approved act of investigating a case of chronic occupational disease being sent?
1. To a person ill or to a person representing his interests.

2. Insurer.
3. To the insurer.
4. To the state labor inspector.
5. Medical institution.

26. What should a district doctor do when he suspects an acute occupational disease?
1. Fill out an emergency notice.
2.Send an emergency notification within 12 hours from the moment of filling in the regional center of occupational pathology.
3. Send an emergency notice within 12 hours from the moment of filling in the Republican Center for Occupational Pathology and Allergology.
4. Send an emergency notice within 12 hours from the moment of filling in the center of hygiene and epidemiology.
5. Immediately send a telephone message or fax to the center of epidemiology, if there are more than 2 people injured with a disability.

27. What should the local doctor indicate in the emergency notification of an acute occupational disease?
1. Diagnosis.
2. Estimated harmful factors of the production process that caused the disease is an acute occupational disease.
3. The alleged causes and circumstances that caused the acute occupational disease.
4. The work of the victim in the enterprise.
5. The list of diseases that were previously in the victim.

28. When should a notice of acute occupational disease be sent immediately by phone or fax to the regional center for hygiene and epidemiology?
1. In cases of acute occupational disease with disability, if two or more people are injured.
2. In case of serious condition of the victim and the need to provide him with specialized medical care.
3. If it is personally required by the victim.
4. In cases of anthrax, brucellosis, tetanus, rabies and other especially dangerous infections associated with the professional activities of the victim.
5. In case of sudden death of the victim.

29. What should the commission do when investigating a case of an acute occupational disease?
1. To examine the workplace in detail with an assessment of the working conditions and production factors that led to an adverse effect on the victim.
2. Identify the causes of acute professional illness or poisoning.
3. To establish persons involved in the occurrence of an acute occupational disease.
4. To study the effectiveness of measures to eliminate the causes and prevent similar diseases or poisoning in the future.
5. To determine the degree of guilt of the administration of the enterprise in the occurrence of an acute occupational disease.

30. Which of the following refers to the medical rehabilitation of persons with occupational diseases?

1.Timely, as early as possible detection of working-ing initial clinical symptoms, both occupational pathology and non-professional diseases.
2. Purpose of pathogenetically substantiated rehabilitation treatment for victims of harmful factors of the working environment - drug and non-drug (physiotherapy, physiotherapy, spa treatment).
3. Temporary employment of persons with clinical manifestations of occupational pathology.
4. Establishment of a medical rehabilitation rehabilitation expert commission (MEDC) of the 3rd disability group for the period of acquiring a new specialty
5. Rational employment of persons with disabilities in groups II and I (work in specially created conditions, including at home).

31. Which of the following refers to the labor reabilitation of persons with occupational diseases?
1.Timely, as early as possible detection of working-ing initial clinical symptoms, both occupational pathology and non-professional diseases.
2. Purpose of pathogenetically substantiated rehabilitation treatment for victims of harmful factors of the working environment - drug and non-drug (physiotherapy, physiotherapy, spa treatment).
3. Temporary employment of persons with clinical manifestations of occupational pathology.
4. Establishment of the MEDICAL III disability group for the period of acquiring a new specialty.
5. Rational employment of persons with disabilities in groups II and I (work in specially created conditions, including at home).

32. Which of the following refers to the social and labor rehabilitation of disabled people with occupational diseases?
1.Timely, as early as possible detection of working-ing initial clinical symptoms, both occupational pathology and non-professional diseases.
2. Purpose of pathogenetically substantiated rehabilitation treatment for victims of harmful factors of the working environment - drug and non-drug (physiotherapy, physiotherapy, spa treatment).
3. Temporary employment of persons with clinical manifestations of occupational pathology.
4. Establishment of the MEDICAL III disability group for the period of acquiring a new specialty.
5. Rational employment of persons with disabilities in groups II and I (work in specially created conditions, including at home).

33. What are the functions of specialized professional pathological MEDC?
1. Determination of the degree of loss of professional working ability.
2. Determination of additional types of damage compensation: expenses for sanatorium-resort treatment, prosthetics, additional nutrition
3. The establishment of the maximum amount of monetary compensation in connection with disability.
4. Determination of the list of free medicines that the victim needs.
5. Determination of need for outside care.

34. In which cases the patient should be sent for examination at the MEDNC?

1. When identifying signs of persistent partial or complete loss of employment.
2. When temporary disability, if the sick-list should be extended continuously for a period exceeding 2 months.
3. In case of temporary disability, if the sick leave has to be extended continuously for a period exceeding 4 months.
4. When temporary disability, if the sick-list should be extended with a break for a period exceeding 5 months.
5. When temporary disability, if the sick-list should be extended with a break for a period exceeding 6 months.

35. What functional class (FC) of the state of the functions and vital activity of the organism corresponds to the absence of impairment of function and limitation of vital activity?
1.FC-0.
2. FC-1.
3. FC-2.
4. FC-3.
5. FC-4.

36. What functional class of the state of the functions and vital activity of the organism corresponds to a light restriction of vital activity due to the loss of up to 25% of the functions?
1.FC-0.
2. FC-1.
3. FC-2.
4. FC-3.
5. FC-4.

*37. What functional class of the state of functions and vital activity of the organism corresponds to a moderate limitation of vital activity due to the loss of 26 to 50% of the functions***?**
1.FC-0.
2. FC-1.
3. FC-2.
4. FC-3.
5. FC-4.

38. What functional class of the state of functions and vital activity of the organism corresponds to a significant limitation of vital activity due to the loss of 51 to 75% of the functions?
1.FC-0.
2. FC-1.
3. FC-2.
4. FC-3.
5. FC-4.

39. What functional class of the state of functions and vital activity of the organism corresponds to a sharp disturbance, with marked limitation of vital activity up to the complete loss of functions - 76-100%?
1.FC-0.

2. FC-1.
3. FC-2.
4. FC-3.
5. FC-4.

40. Which definitions correspond to the third group of disability?
1. Complete loss of working ability and the patient needs constant assistance, care or supervision.
2. Labor is not available (due to pronounced functional limitations caused by the disease).
3. Labor is contraindicated (due to the aggravation of the patient's condition as a result of any professional work activity).
4. Forced transition for lower health status due to health reasons (decrease in qualification by 4 grades or more, reduction of category, management level for managers).
5. Reduction due to health status of norms, the volume of production, reduction for the same reasons, the duration of the working day.

41. What are the definitions correspond to group II disability?
1. Complete loss of working ability and the patient needs constant assistance, care or supervision.
2. Labor is not available (due to pronounced functional limitations caused by the disease).
3. Labor is contraindicated (due to the aggravation of the patient's condition as a result of any professional work activity).
4. Forced transition for lower health status due to health reasons (decrease in qualification by 4 grades or more, reduction of category, management level for managers).
5. Reduction due to health status of norms, the volume of production, reduction for the same reasons, the duration of the working day.

42. Which definitions correspond to the group I disability?
1. Complete loss of working ability and the patient needs constant assistance, care or supervision.
2. Labor is not available (due to pronounced functional limitations caused by the disease).
3. Labor is contraindicated (due to the aggravation of the patient's condition as a result of any professional work activity).
4. Forced transition for lower health status due to health reasons (decrease in qualification by 4 grades or more, reduction of category, management level for managers).
5. Reduction due to health status of norms, the volume of production, reduction for the same reasons, the duration of the working day.

43. To whom is a group of disability due to occupational disease established indefinitely?
1. Disabled men over 55 years old and disabled women over 50 years old.
2. Disabled men over 60 years old and disabled women over 55 years old.
3. Invalids of groups I and II for men and women who have a disability group for 15 years have not changed or a higher group of disabilities has been established.

4. Men on reaching the age of 55 and women 50 years of age who continuously up to this age had group I disability for the last 5 years.
5. Invalids with severe forms of diseases characterized by persistent and irreversible changes in internal organs.

44. What are the benefits granted to persons suffering from occupational diseases?
1.Payment of 100% of the average salary for a sick leave.
2. The size of the disability pension does not depend on the length of service in harmful conditions.
3.Free vouchers to the sanatorium.
4. Monetary compensation of the employer's proven guilt in the occurrence of occupational disease.
5. The pre-emptive right in obtaining state living space.

45. On whose initiative are preliminary inspections of persons taking jobs at work with the presence of occupational hazards?
1. Trade unions.
2. The employer.
3. Local health authorities.
4. Personal initiative applying for a job.
5. Employment services.

46. What document is issued to a person who has undergone a preliminary inspection before applying for work in hazardous conditions?
1.Help indicating "fit" to work in harmful conditions.
2. Help with the indication "not suitable" to work in harmful conditions.
3.Medical conclusion indicating the causes of unsuitability, if any.
4.Act of a medical examination with a detailed description of the results of clinical and laboratory research and a reasonable conclusion - fit or not fit for work.
5.No any documents on the hands of those who have passed the preliminary axis are not issued.

47. What document is issued as a result of periodic medical examination of persons working in hazardous conditions?
1.Help indicating "fit" to work in harmful conditions.
2. Help with the indication "not suitable" to work in harmful conditions.
3. Act of periodic medical examination.
4. Act of medical examination with a conclusion: a general non-professional disease was revealed, indicating the diagnosis and recommendations for treatment and rational employment.
5. Act of medical examination with a conclusion: an occupational disease is suspected, additional examination or dynamic observation of a shop doctor is required.
48. What recommendations does the commission give in the final act of a periodic medical examination in identifying a common, non-professional disease?
1. An employee can continue professional work
2. The worker is to be treated.
3. The worker is subject to follow-up.
4. Further work in contact with occupational hazards is contraindicated (VKK gives recommendations on transferring to another job).

5. The worker is subject to dismissal for health reasons.

49. Who is subject to periodic medical examinations?
1. Persons working with harmful and hazardous substances and under the influence of adverse production factors.
2. Working pensioners.
3.Repairs and installers in enterprises with harmful and hazardous working conditions.
4. Seasonal workers.
5. Workers at hazardous and hazardous facilities.

50. Who is contraindicated in employment and work in conditions of adverse effects of factors of the working environment?
1. Persons suffering from chronic diseases with violations of the functions of internal organs.
2. Pregnant women.
3. For women during lactation.
4. Persons of both sexes over 50 years old.
5. To persons under 18 years of age to work under the ground and associated with high physical stress.

Section II
PROFESSIONAL DISEASES OF RESPIRATORY ORGANS CAUSED BY EXPOSURE TO INDUSTRIAL AEROSOLS

Control tests to section II

1. Which diseases listed below are not caused by inhalation of industrial dust?
1. Pneumoconiosis.
2. Byssinosis.
3. Chronic bronchitis.
4. Pneumonia.
5. Bronchial asthma.

2. What dust particles are deposited on the mucous membrane of the trachea and large bronchi?
1. The sizes are more than 20 microns.
2. The sizes are more than 10 microns.
3. The sizes are less than 5-7 microns.
4. All sizes mentioned.
5.None of the above.

3. What dust particles are deposited on the mucous membrane of the middle and small bronchi?
1. The sizes are more than 20 microns.
2. The sizes are more than 10 microns.
3. The sizes are less than 5-7 microns.
4. All sizes mentioned.
5.None of the above.

4. What dust particles enter the lumen of the alveoli?
1. The sizes are more than 20 microns.
2. The sizes are more than 10 microns.
3. The sizes are less than 5-7 microns.
4. All sizes mentioned.
5.None of the above.

5. Dust particles, what sizes are deposited in normal alveoli?
1. The sizes are more than 20 microns.
2. The sizes are more than 10 microns.
3. The sizes are less than 5-7 microns.
4. All sizes mentioned.
5.None of the above.

6. Inhalation of which dust causes silicosis?
1. Containing silica particles.
2. Containing particles of silicon hydroxide.
3. Containing asbestos particles.
4. Containing talc particles.
5. Containing apatite particles.

7. *Inhalation of which dust causes the formation of silicatosis?*
1. Containing silica particles.
2. Containing particles of silicon hydroxide.
3. Containing particles of metals and their compounds.
4.Containing carbon particles.
5. Containing particles of organic compounds.

8. Inhalation of what dust causes the formation of carboconiosis?
1.Containing quartz particles.
2.Containing asbestos particles.
3. Containing talc particles.
4. Containing apatite particles.
5. Containing particles of coal, graphite.

9. Inhalation of what kind of dust causes the formation of metal consoles?
1.Containing quartz particles.
2.Containing asbestos particles.
3.Containing iron particles.
4.Containing graphite particles.
5.Containing talc particles.

10. What does the fibrogenicity (ability to cause pulmonary fibrosis) of industrial dust primarily depend on?
1. From the concentration of dust particles in the air we breathe.
2. From the content in the dust particles of silicon dioxide.
3. From the content of silicon hydroxide particles in dust.
4. From the content in the dust of fungi and bacteria.
5. From the duration of stay in dusty conditions.

11. What kind of dust cause silicosis?
1. Highly fibrogenic, containing more than 10% of silicon dioxide.
2. Weak-fibrogenic, containing less than 10% silica.
3.Afibrogenic, not containing silicon dioxide, with toxic-allergic properties.
4. All listed.
5.None of the above.

12. What dusts cause silicates?
1. Highly fibrogenic, containing more than 10% of silicon dioxide.
2. Weak-fibrogenic, containing less than 10% silica.
3.Afibrogenic, not containing silicon dioxide, with toxic-allergic properties.
4. All listed.
5.None of the above.

13. What features are not typical for silicosis?
1. Long-term inhalation of dust containing more than 10% freedom of silica.
2. Formation in the lung nodular interstitial fibrosis.
3. Formation in the interstitial tissue of the lung granulomas from epithelioid cells.
4. The defeat of the bronchial tree in the form of peribronchitis.
5. Violation of the ventilation function of the lungs mainly by restrictive type.

14. What features are typical for silicosis?
1. Long-term inhalation of dust containing less than 10% silica.
2.In the interstitial lung tissue granulomas are formed mainly from macrophages.
3. The defeat of the bronchial tree in the form of endobronchitis.
4. Violation of the ventilation function of the lungs mainly by obstructive type.
5. All are typical.

15. Where do silicotic nodules form in the lungs?
1. In the wall of the alveoli and alveolar passages.
2.In peribronchial and perivascular interstitial spaces.
3. In the course of the lymphatic vessels.
4. In all specified places.
5.No in one of these places.

16. What can occur on the site of silicotic nodules?
1. Drain fields of fibrosis.
2. Lime deposits.
3. Caverns.
4. All of the above.
5. None of the above.

17. What kind of silicosis is, formed during the year of work in the environment of highly fibrogenic dust (containing more than 10% SiO2), characterized by persistently progressive flow, rapid, within 1-3 years, the formation of massive fibrosis?
1. **To "acute" silicosis.**
2. To chronic rapidly progressive silicosis.
3. To chronic slowly progressive silicosis.
4. To chronic late silicosis.
5.No to one of the above.

18. What kind of silicosis, formed during 3-5 years of work in an environment of moderately fibrogenic dust (containing less than 10% SiO2), characterized by a progressive course, with transitions from the initial to the subsequent stages of the disease after 2- 3 years?
1. To "acute" silicosis.
2. To chronic rapidly progressive silicosis.
3. To chronic slowly progressive silicosis.
4. To chronic late silicosis.
5.No to one of the above.

19. What kind of silicosis, formed over 10-12 years of work in an environment of weakly fibrogenic dust, characterized by latent onset, transitions from the initial to the subsequent stages of the disease in 5-10 years?
1. To "acute" silicosis.
2. To chronic rapidly progressive silicosis.
3. To chronic slowly progressive silicosis.
4. To chronic late silicosis.
5.No to one of the above.

20. What type of silicosis is, which arose several years after the cessation of contact with fibrogenic dust, characterized by continuously progressive pulmonary fibrosis?
1.K "acute" silicosis.
2. To chronic rapidly progressive silicosis.
3. To chronic slowly progressive silicosis.
4. To chronic late silicosis.
5.No to one of the above.

21. What is the type of pneumoconiosis that has arisen as a result of inhalation of dust containing particles of graphite, iron, hydro-silicon oxide?
1.K "acute" silicosis.
2. To chronic rapidly progressive silicosis.
3. To chronic slowly progressive silicosis.
4. To chronic late silicosis.
5.No to one of the above.

22. What stage or form of silicosis corresponds to the X-ray-logical picture of bilateral amplification, deformation of the broncholecular pattern, symmetrical expansion, compaction, deformation of the roots of the lungs, absence of expansion of the shadow of the heart?
1. Silicosis of the I stage.
2. Silicosis of stage II.
3. Silicosis of stage III.
4. Silicon tuberculosis.
5. Kaplan syndrome.

23. What stage or form of silicosis corresponds to the X-ray-logical picture of compaction, expansion, deformation of the light roots, strengthening of the bronchopulmonary pattern, basal emphysema, thickening, deformation of the pleural contours in combination with nodular changes, symmetrically scattered throughout all fields, having the same dimensions (from 1-2 to 18-10 mm) and density?
1. Silicosis of the I stage.
2. Silicosis of stage II.
3. Silicosis of stage III.
4. Silicon tuberculosis.
5. Kaplan syndrome.

24. What stage or form of silicosis corresponds to an X-ray logical pattern of asymmetrically located large nodes of various sizes, a form against the background of a rough deformation of the bronchopulmonary pattern, roots of the lungs, thickening and deformity of the pleura, interpleural adhesions, bullous emphysema, -samples of slit-like, non-level liquid ca-vern?
1. Silicosis of the I stage.
2. Silicosis of stage II.
3. Silicosis of stage III.
4. Silicotuberculosis.
5. Kaplan syndrome.

25. What complications are typical for silicosis?
1. Chronic dust bronchitis.

2. Emphysema of the lungs.
3. Pleurisy.
4. Pulmonary heart.
5. All are typical.

26. What are the clinical symptoms that usually appear during the reversal of silicosis to silicotuberculosis?
1. Detection of mycobacterium tuberculosis in sputum.
2. Blood clots.
3. Subfebrile condition.
4. All listed.
5. None of the above.

27. What are the clinical symptoms that usually appear when reversing silicosis to silicotuberculosis?
1. A cough arises or intensifies.
2. Sweating.
3. Positive results of skin tuberculin tests.
4. All listed.
5. None of the above.

28. What radiological signs can indicate the transition of silicosis to silicotuberculosis?
1. Shell-shaped calcification of mediastinal lymph nodes.
2.Polymorphic dissemination foci, located mainly in the upper parts of the lungs.
3. Rounded cloudy segmental infiltrates, sometimes occupying a whole lobe of the lung.
4. All listed.
5. None of the above.

29. What are the typical deviations for silicoarthritis?
1. Diffuse interstitial or nodular radiological changes in the lungs.
2. Round infiltrative formations with sizes from 0.5 to 2 cm in diameter in the peripheral regions of the lungs.
3. Erosive ankylosing symmetric arthritis of the small joints of the hands.
4. All listed.
5. None of the listed deviations.

30. In what cases arise silicates?
1.When inhalation of multicomponent silicate dust containing up to 10% silica.
2.When inhaled, multicomponent silicate dust containing more than 10% silica.
3. When ingested into the digestive tract of dust particles containing a large amount of free silicon dioxide.
4. In all listed cases.
5.No in one of the listed cases.

31. Inhalation of dust, which of the following minerals can lead to the occurrence of pneumoconiosis - silicatosis?
1. Asbest.
2. Talc.
3. Mica.

4. All listed.
5. None of the above.

32. Which of the following is not typical for silicosis - asbestos?
1. Diffuse fibrosis of the lungs with the involvement of peribronchial, perivascular interstitial tissue, interlobular and alveolar-dividing partitions.
2. Formation of bronchiectasis.
3. Accumulation of asbestos dust particles in bifurcation and basal lymph nodes.
4. Occurrence in the lungs symmetrically located nodules.
5.High risk of squamous cell carcinoma from the epithelium of the mucous membrane of the small bronchi.

33. What clinical manifestations are not typical for silicatosis - asbestosis?
1. The combination of symptoms of chronic dust bronchitis and pulmonary fibrosis.
2. Warts on the skin.
3. Linear microstructures with thick ends in the form of weights, drum sticks in the histological preparations of the lungs.
4. Thick, viscous, difficult to discharge sputum.
5. Hemoptysis

34. What allows you to confirm the diagnosis of silicatosis - asbestos?
1. The professional route confirms long-term work in industries related to the extraction, processing, use of asbestos in the production of the mineral.
2. The results of the hygienic examination confirming the presence of excess of the maximum permissible concentrations of asbestos dust in the air at the workplace of the sick person.
3. Characteristic clinical manifestations of the disease, detection of asbestos fibers and bodies in the sputum.
4.Results of studies of respiratory function, X-ray, ECG, other methods confirming the presence of changes in the lung structure, respiratory insufficiency, signs of pulmonary heart characteristic of asbestosis.
5. The aggregate of all of the above.

35. What changes are not typical for silicosis - talcosis?
1. The combination of chronic dust bronchitis with pulmonary emphysema.
2. Miliary interstitial pulmonary fibrosis.
3. Formation in light nodules.
4. Merge nodules in large nodes.
5. The accumulation of dust particles in the basal lymph nodes.

36. What changes are not typical for pneumoconiosis - anthracosis?
1.In the lungs there are anthracotic foci that can merge into large fields of fibrosis.
2. Formation of cavities in the foci of pulmonary fibrosis.
3. Presence of more than 10% silica in coal dust.
4. All of these shifts are typical.
5. The indicated shifts are not typical for anthracosis.

37. What are the typical shifts for pneumoconiosis - anthracosis?
1. The combination of chronic dust bronchitis with pulmonary emphysema.

2. Miliary interstitial pulmonary fibrosis.
3. Formation in light nodules.
4. Merge nodules in large nodes.
5. The accumulation of dust particles in the basal lymph nodes.

38. What radiological changes in the lungs are typical for metallodiosis - siderosis?
1. Small radiopaque foci scattered throughout all pulmonary fields with distinct, uneven contours/
2. Caverns.
3. Bronchiectasis.
4. All are typical.
5. Everything is not typical.

39. What type of pneumoconiosis is berylliosis?
1. Pneumoconiosis caused by a highly fibrogenic agent.
2. Pneumoconiosis caused by a weakly fibrogenic agent.
3. Pneumoconiosis caused by a toxic-allergic agent.
4. All definitions are correct.
5. All definitions are incorrect.

40. What features are not typical for berylliosis?
1.Beryllium can be deposited for a long time in braids and lung tissue, replacing magnesium there.
2. The disease may develop by inhaling minimal concentrations of beryllium dust in a very short time.
3. For the formation of the disease requires a long, many years of contact with the dust of metallic beryllium or its alloys.
4. Toxic doses of beryllium are able to penetrate the body through intact skin.
5. All are typical.

41. What pathological changes are typical for berylliosis?
1. Formation of hypersensitive immune complex pneumonitis.
2. Formation of diffuse granulomatous pulmonary fibrosis.
3. Formation of granulomas in the internal organs.
4. All are typical.
5. Everything is not typical.

42. What features are not typical for beryllium granulomatous lesions of the lungs?
1.Berylliotic granulomas are formed by epithelioid cells.
2. In the central part of the granuloma are located the cochlear bodies.
3.Beriliosis granulomas can merge into large nodes.
4. In granulomas may occur foci of caseous necrosis.
5. All are typical.

43. What clinical forms are typical for acute berylliosis (inhalation of beryllium aerosols)?
1. Acute tracheobronchitis.
2. Acute broncho-bronchiolitis.
3. Toxic pneumonitis.
4. All are typical.

5. Everything is not typical.

44. What are the clinical features are not typical for chronic berylliosis?
1. The disease is formed 1-2 years after the first contact with beryllium.
2. The disease begins with shortness of breath, gradually turning into asphyxiation.
3. During coughing episodes, blood streaks appear in the abundant sputum.
4.Board pain in the chest.
5. The recurrent fever is disturbing.

45. What are the objective symptoms typical of chronic berylliosis?
1. Diffuse warm cyanosis.
2. The end of the phalanges of the fingers in the form of "drum sticks", the nail - "watch glasses".
3. In the lungs, non-sound crepitations (“crackling of cellophane”), dry and finely bubbling moist rales are heard.
4. All are typical.
5. Everything is not typical.

46. What radiographic changes in the lungs are not typical for chronic berylliosis?
1. Interstitial changes in the form of reinforcement, deformity of the congestive and bronchopulmonary patterns.
2. Drain granulomatous changes in the form of small-point nodules, compaction, expansion of the roots of the lungs.
3. Cloudy shadows of infiltrates occupying the apical regions of the right and left lungs.
4. All are typical.
5. Everything is not typical.

47. Which of the following occupational diseases relate to mixed dust pneumoconiosis?
1. Siderosilicosis.
2. Silikoantrakoz.
3. Pneumoconiosis of electric welders.
4. All mentioned.
5. None of the above.

48. What are the components of the welding gas-aerosol mixture cause pulmonary fibrosis when the pneumoconiosis of welders occurs?
1. Iron aerosol.
2. Aerosol of beryllium.
3. Silicon dioxide aerosol.
4. Carbon monoxide.
5. Suspended soot particles.

49. What are the components of the welding gas-aerosol mixture cause toxic-allergic pneumonitis, diffuse granulomatous pneumofibrosis in case of pneumoconiosis of welders?
1. Iron aerosol.
2. Aerosol of beryllium.
3. Silicon dioxide aerosol.

4. Carbon monoxide.
5. Suspended soot particles.

50. What are the components of the welding gas-aerosol mixture can cause the occurrence in patients with pneumoconiosis of welders of professional asthma?
1. Iron aerosol.
2. Silicon dioxide aerosol.
3. Chromium aerosol.
4. Carbon monoxide.
5. Suspended soot particles.

51. Which of the following diseases do not belong to pneumoconiosis?
1. Silicosis
2. Asbestosis.
3. Antraköz.
4. Byssinosis.
5. Farmer's lung.

52. What caused the damaging effect of fibrous dust that causes the formation of byssinosis?
1. By the presence on the surface of fibers of molds, producing histamine-like substances.
2. By the presence in the composition of cotton fibers of substances capable of inactivating histaminase in the bronchial mucosa.
3. Sensitizing properties of protein substances that are part of the plant fibers.
4. All of the above.
5. None of the above.

53. What are the clinical manifestations are not typical for byssinosis?
1. Dry cough 1-2 hours after starting to work in dusty conditions.
2. After the weekend work in the dust causes attacks of strangling.
3.During asthma attacks, body temperature may rise.
4. During the working week, the severity of bronchospastic phenomena gradually subsides.
5. All are typical.

54. What definitions do not apply to chronic occupational dust bronchitis?
1. Chronic diffuse non-allergic inflammatory disease of the bronchi.
2. The disease occurs as a result of long-term work in conditions of high content of moderately aggressive mixed dust in the inhaled air.
3. The disease leads to a progressive impairment of pulmonary ventilation and gas exchange of the obstructive type.
4. The disease can precede or be combined with occupational bronchial asthma.
5. All are related.

55. What are the features of the pathogenesis of chronic occupational dust bronchitis?
1. Endobronchitis arises and gradually progresses, which can lead to irreversible obstruction of the bronchi.
2. A nonspecific hyperreactivity of the bronchial muscles arises, which manifests itself as a bronchospastic response in response to the impact of the dust factor.

3. Diffuse granulomatous pneumosclerosis occurs, resulting in restrictive disorders of the ventilating function of the lungs.
4. All are related.
5. All do not apply.

56. What are the clinical manifestations of the first stage of chronic occupational dust bronchitis?
1. Non-obstructive respiratory failure (DN0 - DNI).
2. Respiratory failure DNI-DNII obstructive or mixed type.
3. Respiratory failure DNII-DNIII of mixed obstructive-restrictive type.
4. Lesion of the mucous membrane of the large bronchi (endobronchitis irritation).
5. Deforming bronchitis with the defeat of all layers of the wall.

57. What are the clinical manifestations of the second stage of chronic occupational dust bronchitis?
1. Non-obstructive respiratory failure (DN0 - DNI).
2. Respiratory failure DNI-DNII obstructive or mixed type.
3. Respiratory failure DNII-DNIII of mixed obstructive-restrictive type.
4. Lesion of the mucous membrane of the large bronchi (endobronchitis irritation).
5. Deforming bronchitis with the defeat of all layers of the wall.

58. What are the clinical manifestations of the third stage of chronic occupational dust bronchitis?
1. Non-obstructive respiratory failure (DN0 - DNI).
2. Respiratory failure DNI-DNII obstructive or mixed type.
3. Respiratory failure DNII-DNIII of mixed obstructive-restrictive type.
4. Lesion of the mucous membrane of the large bronchi (endobronchitis irritation).
5. Deforming bronchitis with the defeat of all layers of the wall.

59. Which wording is closest to the definition of occupational bronchial asthma?
1. A disease characterized by chronic inflammation of the airways that occurs in response to contact with certain chemicals in production. It is determined by the presence of a history of respiratory symptoms, such as wheezing, shortness of breath, feeling of congestion in the chest and cough, the severity of which changes with time, as well as the variability in limiting the speed of air flow.
2. Chronic immuno-inflammatory disease of the broncho-lung system that occurs in conditions of exposure to the organs of respiration of toxic-chemical agents.
3. Chronic occupational respiratory disease, manifested by nonspecific hyperresponsiveness of the bronchopulmonary system in response to exposure to aerosols and dust of certain chemical substances present in the workplace of the patient.
4. All correspond to the definition of occupational bronchial asthma.
5.None of the wording is consistent with this definition.

60. Which allergens are most often the cause of the formation of occupational bronchial asthma?
1. High-molecular substances (molecular weight over 500 daltons) of animal and vegetable origin, occurring under production conditions.
2. Low molecular weight substances (molecular weight less than 500 daltons) used in the chemical and pharmaceutical industry.

3. Pollen of flowering cereals in spring and summer.
4. The saliva of bloodsucking insects.
5. Food allergens (contact with them outside the production process).

61. What circumstances are typical for occupational bronchial asthma?
1.Hypersensitivity type I (atopic) to any allergen present in the workplace of the patient.
2. The occurrence in the workplace of attacks of dry cough, ex-pyratory dyspnea, attacks of breathlessness by inhalation of air containing allergens with a gradual improvement in the state at the end of the shift.
3. Prone to asphyxiation starts to disturb the patient only at the end of the work shift or when he returns home, often at night.
4. All are typical.
5. Everything is not typical.

62. What medicines are used for stopping **the attacks of bronchospasm in patients with occupational bronchial asthma?**
1. β2-agonists of short action (salbutamol, fenoterol).
2.Anticholinergic drugs (Ipratropium bromide, Tiotropium bromide, Oxytropium bromide).
3. Inhalation glucocorticosteroids (beclamethasone, budeso-nid, flunisolid, fluticasone, etc.).
4. Theophylline short-acting (aminophylline, aminophylline).
5. Cromoglycate sodium (Intal, cromoline).

63. What medicines are used to treat patients with occupational bronchial asthma during remission?
1. β2-agonists of short action (salbutamol, fenoterol).
2.Anticholinergic drugs (Ipratropium bromide, Tiotropium bromide, Oxytropium bromide).
3. Inhalation glucocorticosteroids (beclamethasone, budeso-nid, flunisolid, fluticasone, etc.).
4. Theophylline short-acting (aminophylline, aminophylline).
5. Antagonists of leukotriene receptors (montelukast).

Section III
PROFESSIONAL DISEASES, CAUSED BY EXPOSURE TO PHYSICAL FACTORS

Control tests to section III

1. What vibration velocity corresponds to the magnitude of the vibration sensation threshold?
1. Less than 10-4 m / s.
2.10-4 m / s.
3. Less than 1 m / s.
4.1 m / s
5. More than 1 m / s.

2. What vibration velocity corresponds to the threshold of pain sensations caused by vibration?
1. Less than 10-4 m / s.
2.10-4 m / s.
3. Less than 1 m / s.
4.1 m / s
5. More than 1 m / s.

3. Vibrations with what frequency are felt tactile and not perceived by the sound analyzer?
1. Less than 16 Hertz (Hz).
2. From 16 to 20,000 Hz.
3. More than 20,000 Hz.
4. With any frequency mentioned.
5.No in one of the mentioned frequency ranges.

4. What frequency ranges are taken into account in the overall effect of vibration?
1.1-4 Hz.
2.8-16 Hz.
3.31.5-63 Hz.
4.125-1000 Hz.
5. All mentioned.

5. What frequency ranges are taken into account when local vibration is applied?
1.1-4 Hz.
2.8-16 Hz.
3.31.5-63 Hz.
4.125-1000 Hz.
5. All mentioned.

6. How does the propagation zone and penetrating ability of vibrations change with an increase in their frequency?
1. The distribution area is increasing.
2. Penetration ability increases
3. The distribution area is reduced.
4. Penetration ability decreases.

5. The penetrating ability and the zone of distribution do not change.

7. What structures are the conductors of vibrational vibrations propagating in the human body?
1. Bones of the skeleton.
2. Muscles.
3. Links.
4. Cartilage structures.
5. Blood vessels.

8. What tissue structures contribute to the extinction of vibrations?
1. Bones of the skeleton.
2. Muscles.
3. Links.
4. Cartilage structures.
5. Blood vessels.

9. What changes in the human body occur when the vibration with a velocity of more than 1 m / s?
1. Injuries in the form of tears of the skin, muscles, internal organs.
2. Disorders of neurohumoral regulation of vascular tone.
3. Violations of the neurohumoral regulation of muscle tone.
4. Atrophy of receptor structures and nerve endings in the system of perception and control of pain, tactile sensations.
5. Pathological changes in the centers of regulation of pain and sensitivity.

10. What changes in the human body can not occur under the action of vibration with a vibration velocity of less than 1 m / s?
1. Violations of the neurohumoral regulation of vascular and muscle tone.
2. Atrophy of receptor structures and nerve endings in the system of perception and control of pain, tactile sensations.
3. Injuries in the form of tears of the skin, muscles, internal organs.
4. Pathological changes in the centers of regulation of pain and sensitivity.
5. Changes in the joints of the limbs and spine (osteoarthritis).

11. What pathogenetic features are typical for local vibration exposure with a frequency of 100-250 Hz?
1. Promotes an increase in volumetric blood flow in the vessels.
2. Causes compensatory activation of vasoconstrictor mechanisms.
3. On weekends, it causes the formation of painful ischemic changes in the limbs subjected to vibration.
4. Contributes to the elimination of ischemia and pain in the limbs when resuming work with a vibrating instrument after a day off.
5. All are typical.

12. What features are not typical for vibration disease from the effects of local vibration?
1. The disease is formed very slowly - for many years working with a vibrating instrument.

2. The disease is formed quickly - within a few months of working with a vibrating instrument.
3. First of all, vibration sensitivity is dulled.
4. All are typical.
5. Everything is not typical.

13. What clinical features are not characteristic of vibration disease from local effects on the hands?
1. Pain in the hands outside of work with a vibrating instrument.
2. Increased sensitivity of hands to cold.
3. The occurrence of acroasphyxia with necrosis of the terminal phalanges of the fingers.
4. All are characteristic.
5. Everything is not typical.

14. Which of the listed syndromes form a clinical picture of a vibration disease from local influence?
1. Angiodystonic syndrome.
2.Anemic syndrome.
3. Vegetosensory neuropathy.
4. Hemolytic syndrome.
5. From all of the above.

15. What syndromes correspond to the I severity of vibration disease from local effects?
1. Peripheral angiodystonic syndrome of the upper extremities with rare angiospasms of the fingers.
2. Syndrome of autonomic-sensory polyneuropathy of the upper extremities.
3. The syndrome of sensory-motor polyneuropathy of the upper extremities.
4. Encephalopolyneuropathy syndrome.
5. Syndrome of polyneuropathy with generalized acroangiospasms.

16. What are the variants of autonomic-sensory polyneuropathy of the upper extremities that can occur in patients with II degree of severity of vibration disease from local exposure?
1. With persistent vegetative-trophic disorders on the hands.
2. With dystrophic disorders of the musculoskeletal system of the arms and shoulder girdle (myopatosis, myofibrosis, periarthrosis, arthrosis).
3. With a neck-humeral plexopathy.
4. With cerebral angiodystonic syndrome.
5. All listed.

17. What syndromes correspond to the III severity of vibration disease from local exposure?
1. Peripheral angiodystonic syndrome of the upper extremities with rare angiospasms of the fingers.
2. Syndrome of autonomic-sensory polyneuropathy of the upper extremities.
3. The syndrome of sensory-motor polyneuropathy of the upper extremities.
4. Encephalopolyneuropathy syndrome.
5. Syndrome of polyneuropathy with generalized acroangiospasms.

18. What syndromes form a clinical picture of a vibration illness from the general vibration?

1. Vegeto-vestibular syndrome.
2. Syndrome of autonomic-sensory polyneuropathy of the extremities.
3. Raynaud's syndrome.
4. **Sjogren's syndrome**
5. Alzheimer's syndrome.

19. What syndromes are involved in the formation of the clinical picture of vibration disease from general vibration of I degree?

1.Central or peripheral angiodystonic syndrome.
2. Vegetative vestibular syndrome.
3. Syndrome of sensory or autonomic-sensory polyneuropathy of the lower extremities.
4. The syndrome of sensory-motor polyneuropathy.
5.Discirculatory encephalopathy syndrome in combination with peripheral polyneuropathy - encephalopolyneuropathy syndrome.

20. What syndromes form a clinical picture of a vibration disease from the general vibration of the II degree?

1. Cerebral-peripheral angiodystonic syndrome.
2. Syndrome of sensory or autonomic-sensory polyneuropathy with polyradiculo-neuropathy syndrome.
3. Syndrome of sensory or autonomic-sensory polyneuropathy with secondary lumbosacral radicular syndrome due to osteochondrosis of the lumbar spine.
4. Syndrome of sensory or vegetative-sensory polyneuropathy with functional disorders of the nervous system - neurasthenia syndrome.
5. All listed.

21. What syndromes and pathological processes are typical for the III degree of severity of vibration disease from general vibration?

1. The syndrome of sensory-motor polyneuropathy.
2. Syndrome of circulatory encephalopathy in combination with peripheral polyneuropathy - encephalopolyneuropathy syndrome.
3. Distributed osteoarthritis of the spine, large joint of the extremities.
4. Everything is not typical.
5. All are typical.

22. What methods should be used to diagnose vibration disease?

1. Palletestiometry.
2. Algesiometry.
3. Cold test.
4. Test with reactive hyperemia.
5. All listed.

23. What criteria should not be used to prove the diagnosis of vibration disease?

1. Professional route, indicating a long stay in a production environment, characterized by local or general vibration exposure.
2. Clinical syndromes typical of vibratory disease.
3. Objective registered signs of reduction in vibration and pain sensitivity.

4. Characteristic of the disease changes in the state of the osteo-articular apparatus, muscular and cardiovascular systems.
5. Everything should be used.

24. What is the maximum permissible level of noise exposure for a person in the workplace?
1.20 dB (dB).
2.40 dB
3.60 dB
4.80 dB
5,100 dB.

25. At what intensity can noise cause pain?
1. Over 40 dB.
2. Over 60 dB.
3. More than 80 dB.
4. Over 100 dB.
5. More than 120 dB.

26. At what intensity can noise destroy the sound-conducting structures of the ear?
1. Over 40 dB.
2. Over 60 dB.
3. More than 80 dB.
4. Over 100 dB.
5. More than 120 dB.

27. What is the hearing loss corresponds to hearing loss I step?
1. Decrease in hearing by 1-10 dB.
2. Decrease in hearing by 11-20 dB.
3. Decrease in hearing by 21-30 dB.
4. Decrease in hearing by 31-45 dB.
5. Decrease in hearing more than on 46 dB.

28. What hearing loss corresponds to hearing loss stage II?
1. Decrease in hearing by 1-10 dB.
2. Decrease in hearing by 11-20 dB.
3. Decrease in hearing by 21-30 dB.
4. Decrease in hearing by 31-45 dB.
5. Decrease in hearing more than on 46 dB.

29. What hearing loss corresponds to hearing loss of the third degree?
1. Decrease in hearing by 1-10 dB.
2. Decrease in hearing by 11-20 dB.
3. Decrease in hearing by 21-30 dB.
4. Decrease in hearing by 31-45 dB.
5. Decrease in hearing more than on 46 dB.

30. What hearing loss corresponds to hearing loss stage IV?
1. Decrease in hearing by 1-10 dB.
2. Decrease in hearing by 11-20 dB.

3. Decrease in hearing by 21-30 dB.
4. Decrease in hearing by 31-45 dB.
5. Decrease in hearing more than on 46 dB.

31. What criteria should be used to confirm the diagnosis of occupational disease from exposure to noise?
1. Professional route confirming the fact of working in conditions of noise exceeding 80 dB for at least 10 years.
2. Clinical manifestations of hearing loss and functional changes in the central nervous system.
3. The results of clinical and audiological studies confirming one or another degree of hearing loss.
4. The absence of other, except professional, causes of hearing loss.
5. All listed.

Section IV
PROFESSIONAL DISEASES CAUSED BY OVERVOLTAGE OF INDIVIDUAL ORGANS AND SYSTEMS

Control tests to section IV

1. What work can cause occupational diseases from functional overvoltage?
1. Lifting, holding on weight, moving heavy and oversized objects.
2. Work in conditions of forced, non-physiological posture with excessive flexion, extension, rotation, displacement of the center of gravity of the body.
3. Stereotyped, repeatedly repeated movements.
4. Physical work that does not correspond to the physical development of the worker.
5. Heavy physical labor during the whole working day.

2. What muscle groups are affected by occupational myofibrosis?
1. Muscles providing stereotypical movements.
2. Muscles that support the maintenance of a forced posture.
3. Muscles that ensure the retention of massive objects on weight.
4. None of the above.
5. All listed.

3. What pathological processes are typical for professional myofibrosis?
1. Formation of multiple areas of muscle hyper-irritability in the form of painful or latent trigger points.
2. Activation of trigger points with additional muscle ischemia with the appearance of pain, spontaneous contractions of individual groups of muscle fibers.
3. Massive muscle necrosis ("positional compression" syndrome).
4. Disruption of bone tendons.
5. All are typical.

4*. What pathological processes are typical for professional myofibrosis?*
1. Neuromuscular dysfunction, in the form of spontaneous contractions of individual groups of muscle fibers.
2. Active ischemic fibrogenesis.
3. Sealing muscles, reducing their contractility.
4. All are typical.
5. Everything is not typical.

5. What biochemical test can be used for professional myofibrosis object imaging?
1. Detection of free hemoglobin in the blood.
2. Detection of myoglobin in the blood.
3. Detection of high activity of creatine phosphokinase in the blood.
4. Detection of autoantibodies to myocytes.
5. All listed.

6. Where do professional tendovaginis (tendinitis, tendosynovitis) most often develop?
1.In the distal tendon of the flexor of the forearm.
2. In the distal tendon extensor extensors of the forearm.
3. In the tendon sheaths of the biceps muscles.

4. In tendon sheaths of short shoulder rotators.
5. In all of the above.

7. Under what circumstances do professional tendovaginitis (tendinitis, tendinovi) are formed?
1. In the conditions of prolonged physical overvoltage with static loads arising when weights are kept on weight.
2.When dynamic overloads associated with the rapid movement of objects.
3. At long work with the vibrating tool.
4. In all the circumstances listed.
5.Not under one of the listed circumstances.

8. What pathological processes are typical for professional tendovaginitis (tendinitis, tendosynovitis)?
1. Permanent microtrauma of tendons and tendon sheaths.
2. Overextension of insufficiently vascularized su-hozhyly tissue and tendon sheaths.
3.Ochaceous necrosis of individual fibrous bundles of tendons and tendon sheaths with their subsequent hyalinization and calcification.
4. The transition of the inflammatory process from the tendons to the articular bags leads to the formation of tendobursites.
5. All are typical.

9. What are the clinical manifestations typical for professional tendovaginitis (tendinitis, tendosynovitis)?
1. Pain in the forearms, hands, occurring during manual physical labor.
2. Limitations due to pain of movements in the wrist joint.
3. Increased body temperature.
4. Spontaneous shoulder fractures.
5. All are typical.

10. What are the objective symptoms typical for professional tendovaginitis (tendinitis, tendosynovit)?
1. Muscle atrophy.
2. Swelling in the area of the affected tendons and their vaginas.
3. Pain points at the sites of attachment of tendons to the bone.
4. Bruising at the sites of attachment of tendons to the bone.
5. All are typical.
6. Everything is not typical.

11. What radiological signs are typical for professional tendovaginitis (tendinitis, tendosynovitis)?
1. The centers of calcification along the affected tendons.
2. Subperiosteal fractures of the radius.
3. Osteophytes in places of fixation of the tendon to the bone.
4. Osteoporosis of the humerus.
5. All are typical.

12. What features are typical for humeroscapular periarotrosis?

1. This is a lesion of the musculoskeletal structures involved in the movements of the shoulder joint.
2. The disease is caused by damage to the tendons and muscles that form the rotator cuff.
3. The disease is caused by the defeat of the hypochondrium, supraspinatus, small round and subscapularis muscles.
4. There is a degenerative and / or compression tendinitis of the supraspinatus muscle in combination with shoulder myofascial pain syndrome.
5. All are typical.

13. What features of the clinical picture are typical for shoulder-blade periarthrosis?
1. The disease often occurs in men.
2. The disease is preceded by trauma to the shoulder girdle.
3. It is characterized by complaints of pain in the back, in the shoulder in the region of the deltoid muscle.
4. All are typical.
5. Everything is not typical.

14. What features of the clinical picture are typical for shoulder-blade periarthrosis?
1. It is possible to limit the rotation and discharge of the shoulder between 60-1200.
2. The initial abduction of the shoulder is painless, further causes pain.
3. Movement in the shoulder joint with active resistance to this movement causes the appearance or intensification of pain.
4. Passive movements in the shoulder joint are limited only slightly, by no more than 100.
5. All are typical.

15. Under what circumstances does professional epicondylosis or shoulder tendonitis develop?
1. When performing work with intense pronation and supinae of her forearm.
2. During the long work with the vibrating tool.
3. When performing work related to extension-flexion in the elbow joint.
4. In the conditions of long-term physical overvoltage with static loads that occur when weights are kept on weight.
5. With dynamic overloads associated with fast movements of objects.

16. What pathological processes are typical for occupational epicondylosis or shoulder tendoperiosteitis?
1. Dystrophic changes of the forearm support tendons at the site of attachment to the lateral epicondyle.
2. Dystrophic changes of the extensors of the hand and fingers at the point of attachment to the lateral num-tip.
3. Dystrophic changes of the brachiocephalic muscle at the point of attachment to the lateral num-tip.
4. All are typical.
5. Everything is not typical.

17. What are the clinical features typical for occupational epicondylosis or shoulder tenodoperiosteitis?

1. Constant aching pain in the elbow joint, aggravated by extension, supination of the forearm.
2. Pains do not allow to hold the brush unbent and clenched into a fist.
3. Difficult to lift and hold heavy objects with an outstretched hand.
4. The initial abduction of the shoulder is painless, further causes pain.
5. Movement in the shoulder joint with active resistance to this movement causes the appearance or intensification of pain.

18. What are the clinical features typical for occupational epicondylosis or shoulder tenodoperiosteitis?
1. It is possible to limit the rotation and discharge of the shoulder between 60-1200.
2. The initial abduction of the shoulder is painless, further causes pain.
3. Palpation determines pain points in the region of the lateral or medial condyles of the shoulder.
4. Movement in the shoulder joint with active resistance to this movement causes the appearance or intensification of pain.
5. Passive movements in the shoulder joint are limited only slightly, by no more than 100.

19. What circumstances underlie the formation of a professional radial styloidosis (de Querven's disease)?
1. Pathological neurodystrophic process in the sixth canal of the tendon retainer of the ulnar wrist extensor, sometimes in combination with compression of the external branch of the ulnar nerve.
2. The disease is formed when performing a stereotyped ulnar discharge of the hand in combination with the abduction of the thumb.
3. The defeat of the tendons and muscles that form the rotator cuff of the shoulder.
4. Degenerative or compression tendinitis of the supraspinatus muscle in combination with brachial myofascial pain syndrome.
5. A pathological process that develops in the first canal under the dorsal ligament, in which the tendons of the short straightener and the long abductor of the thumb pass.

20. What circumstances underlie the formation of professional ulnar styloidosis?
1. Dystrophic changes of the extensors of the hand and fingers at the point of attachment to the lateral num-tip.
2. A pathological neurodystrophic process in the sixth canal of the tendon retainer of the ulnar wrist extensor, sometimes in combination with compression of the external branch of the ulnar nerve.
3. The defeat of the tendons and muscles that form the rotator cuff of the shoulder.
4. The disease occurs when performing stereotypical work associated with the maximum extension and abduction of the brush in the radial direction.
5. A pathological process that develops in the first canal under the dorsal ligament, in which the tendons of the short straightener and the long abductor of the thumb pass.

21. What are the clinical manifestations typical of professional radial styloidosis?
1. Pain in the circumference of the styloid process of the radial bone, irradiating in the forearm.
2. A thickened, diseased ligament is palpated over the styloid process.

3. Painful sensations increase with an ulnar abduction of the hand, abduction and extension of the thumb.
4. The impossibility of information between the tips of the fingers of the fifth and first fingers of the hand.
5. All are typical.

22. What radiographic signs are typical for professional radiation styloidosis?
1. Osteophytes are detected near the styloid process, compaction of the surrounding soft tissues.
2. In the initial period of the disease, a thickening of the soft tissues surrounding the styloid process is recorded.
3. In the late stages of the disease, osteophytes, acid focal osteoporosis of the bone are detected.
4. All are typical.
5. Everything is not typical.

23. What clinical manifestations are typical for professional ulnar styloidosis?
1. Pain syndrome associated with the extension and abstraction of the kitty to the radial side.
2. Pain radiating to the IV and V fingers.
3. Painful swelling, localized near the styloid process.
4. Painful sensations increase with an ulnar abduction of the hand, abduction and extension of the thumb.
5. The impossibility of information between the tips of the fingers of the fifth and first fingers of the hand.

24. What radiographic signs are typical for professional radiation styloidosis?
1. Osteophytes are detected near the styloid process, compaction of the surrounding soft tissues.
2. In the initial period of the disease, a thickening of the soft tissues surrounding the styloid process is recorded.
3. In the late stages of the disease, osteophytes, acid focal osteoporosis of the bone are detected.
4. All are typical.
5. Everything is not typical.

25. What circumstances underlie the formation of the pro-flexor stenosis of the flexor of the fingers ("snap finger")?
1. Performing manual work with a long, significant pressure on the palmar surface of the hands.
2. Dystrophic changes of the extensors of the hand and fingers at the point of attachment to the lateral num-tip.
3. A pathological process that develops in the first canal under the dorsal ligament, in which the tendons of the short straightener and the long abductor of the thumb pass.
4. Performing a stereotypical ulnar lead of the hand in combination with the abduction of the thumb.
5. All the above circumstances.

26. What are the clinical features typical for the first phase of the professional stenosing tenovaginitis flexor tendovaginitis ("snap finger")?

1. Usually affects the fingers on one hand, more often I finger of the right hand.
2. There is pain in the palmar surface of the metacarpophalangeal joint, aggravated by the movement of the finger, pressing on this place.
3. For the first time there is a "snap" of the finger - fixation in the folded position, accompanied by pain.
4. Fixation is eliminated by arbitrary tension of the flexors (active extension), after which the pain disappears.
5. All are typical.

27. What are the clinical features typical for the second phase of professional stenosing flexovagous flexor tendovaginitis ("snap finger")?

1. "Snap" finger becomes frequent, accompanied by a pronounced pain reaction.
2. "Latching" of a finger is not eliminated by arbitrary extensor abbreviations.
3. To eliminate the "snap" the patient has to use the second hand (passive extension).
4. On palpation of the pain point on the flexor tendon of the finger, a tight, painful nodule is revealed.
5. All are typical.

28. What features are typical for the third phase of professional flexor stenosis of the flexor of the fingers ("snapping finger")?

1. Due to severe pains, the "snapping-in" cannot be eliminated, and the finger remains locked in a bent or unbent position.
2. Pain points are localized at the site of detection of a tight, painful nodule on flexor tendons and / or extensors of the affected finger.
3. Radiographically, in the later stages of the disease, consolidation foci on flexor tendons are detected near their fixation to the phalanges of the fingers.
4. All are typical.
5. Everything is not typical.

29. What work processes underlie the formation of a professionally conditioned carpal (carpal) canal syndrome?

1. Stereotypical work associated with the maximum extension and abduction of the brush in the radial direction.
2. Work with intense pronation and supination of the forearm.
3. Long-term intensive work with fingers with constant tension of the muscles of the forearm.
4. All listed processes.
5. None of the listed processes.

30. What pathological processes underlie the professionally conditioned carpal (carpal) canal syndrome?

1. The wrinkling of the transverse ligament of the wrist, which forms in conjunction with the bones of the wrist and the palmar ligament of the carpal canal.
2. Chronic stenotic ligamentitis of the transverse ligament of the wrist.
3. The narrowing of the carpal canal, the compression of the finger flexors and n- passing through it. medianus.

4. All listed processes.
5. None of the listed processes.

31. What are the clinical manifestations typical for a professionally conditioned carpal (carpal) canal syndrome?
1. Numbness, paresthesia, pain in II – III fingers, aggravated by extension of the hand, forced flexion position of the hands.
2. Extension II - III fingers of the hand, causing sharp pain.
3. The pain sensitivity of the skin on II - III fingers is reduced.
4. With percussion of the transverse ligament of the wrist, pain appears in the II-III fingers of the hand.
5. All are typical.

32. What joint damage can occur with professional osteoarthritis?
1. Shoulder.
2. Elbow.
3. Carpal.
4. Hip.
5. Knee.

33. What work processes can cause professional osteoarthritis?
1. Large-amplitude rotational movements, systematic pressure in the region of the corresponding joints, overstrain and their trauma.
2. Long-term intensive work with fingers with constant tension of the muscles of the forearm.
3. Performing manual work with a long, significant pressure on the palmar surface of the hands.
4. Works with dynamic overloads associated with fast movements of objects.
5. All listed.

34. What are the clinical features typical of professional osteoarthritis?
1. The absence of pain joints alone, at night.
2. The appearance of pain in the affected joint with the onset of physical activity ("starting" pain).
3. Sudden painful limitation of movement in the joint caused by hitting between the articular surfaces of the cartilage fragment.
4. All are typical.
5. Everything is not typical.

35. What radiological signs are not typical for professional osteoarthritis?
1. X-ray positive zone of subchondral sclerosis.
2. The flattening of the articular surfaces.
3. Regional osteophytes.
4. Subluxations of the affected joint.
5. Uzury articular surface.

36. What pathological process underlies professional bursitis?
1. Chronic stenotic ligamentitis of the transverse ligament of the wrist.
2. The narrowing of the carpal canal, the compression of the finger flexors and n-passing through it. medianus.

3. Chronic aseptic inflammation of the synovial bags of the joints, resulting from prolonged overstrain and constant trauma of the joints.
4. Focal necrosis of individual fibrous bundles of tendons and tendon sheaths followed by their hyalinization and calcification.
5. All listed.

37. Which bursites are often professionally determined?
1. Elbow.
2. Pretellar.
3. Poddeltoidnye.
4. All mentioned.
5. None of the above.

38. What pathological changes are typical for the formation of professional bursitis?
1. In the cavity and on the wall of the articular bag free bodies of cartilaginous density, calcifications are formed.
2. Forming in the cavity of the articular pseudotumor sac, which limits the mobility of the joint.
3. Violation of the integrity of the articular bag with the occurrence of he-martroses.
4. Formation of purulent inflammatory process, often leading to perforation of the articular sac.
5. All are typical.

39. What professionally conditioned bursitis can be accompanied by restriction of joint mobility?
1. Elbow
2.Preatelary.
3. Poddeltovidnye.
4. All mentioned.
5. None of the above.

40. What are the clinical and anamnestic features are not typical for professionally caused bursitis?
1. Professional bursitis forms quickly, during several months of work with constant traumatization of articular bags.
2. Professional bursitis forms very slowly and can take place only with long working experience.
3. With a strong pressure on the periarticular surface pain occurs.
4. A spherical tumor with fluctuating contents is detected near the joint.

41. What features are typical for professionally determined coordinating neurosis?
1. The reconciliation of the functions of the central and peripheral nervous system, resulting in the loss of a high degree of differentiation acquired as a result of the professional activity of the stereotypical motor stereotype.
2. The disease develops in highly skilled professionals engaged in intensive labor, usually against the background of general neuroticism.
3. The disease can occur in persons whose work is associated with the implementation of highly differentiated movements performed at an accelerated rate.

4. All are typical.
5. Everything is not typical.

42. What features are not typical for a professionally-defined focal neurosis?
1.The ability to perform complex professional motor skills is lost.
2. An attempt to perform highly differentiated movements performed at an accelerated rate provokes the appearance of tremor, cramps, weakness, pain.
3. There are signs of organic damage to the central and peripheral nervous system.
4. The motive acts, not connected with the thin professional movements, are carried out easily and without any restrictions.
5. All are typical.

43. What are the forms of professional coordinator neurosis?
1. Furious.
2. Sensitive (neural).
3.Pareticheskaya.
4. Shuttle.
5. All listed.

44. What form of coordinating neurosis is characterized by non-arbitrary hand trembling while performing thin, differentiated hand movements, which makes writing, playing the piano, typing letters impossible?
1. Furious.
2. Sensitive (neural).
3.Pareticheskaya.
4. Shuttle.
5. For all given.

45. What form of coordinating neurosis is characterized by the appearance of pain when trying to perform coordinated, precise movements with the fingers?
1. Furious.
2. Sensitive (neural).
3.Pareticheskaya.
4. Shuttle.
5. For all given.

46. What form of coordinating neurosis is characterized by the appearance of sudden weakness, lethargy, uncontrollability of fingers when trying to write a text - the pen "falls out" from the hands?
1. Furious.
2. Sensitive (neural).
3.Pareticheskaya.
4. Shuttle.
5. For all given.

47. What form of coordinating neurosis is characterized by a violation of the ability to write - "writing spasm", playing musical instruments?
1. Furious.
2. Sensitive (neural).
3.Pareticheskaya.

4. Shuttle.
5. For all given.

Section V
PROFESSIONAL DISEASES, CAUSED BY EXPOSURE TO CHEMICAL SUBSTANCES

Control tests for Section V

1. Which drugs listed below belong to the group of etiotropic antidotes?
1. Donators of SH-groups - Unithiol, sodium thiosulfate.
2. Cholinesterase reactivators - dipyroxime, isonitrosine, alloxime.
3.Anticholinergic - atropine, ganglioblokatory (pentamine, benzogeksony).
4. Complexes - thetacin-calcium, pentacin, D-penicylamine.
5. All listed.

2. Which drugs listed below belong to the group of antidotes of pathogenetic action?
1.Anticholinergic - atropine, ganglioblokatory (pentamine, benzogeksony).
2. Donators of SH-groups - Unithiol, sodium thiosulfate.
3. Cholinesterase reactivators - dipyroxime, isonitrosine, alloxime.
4. Complexes - thetacin-calcium, pentacin, D-penicylamine.
5. All listed.

3. Which of the following is included in the complex of measures performed in the treatment of acute and chronic intoxications that have arisen in an industrial environment?
1. Immediate cessation of contact with the toxic substance by removing the toxin from the affected area, cleansing the respiratory tract, skin, mucous membranes, digestive tract from the toxic substance.
2. Activation of the intensive elimination of the toxic substance already in the blood and in the depot.
3. Introducing antidotes to neutralize toxins in the body.
4. Restoration of the functions of internal organs disturbed by the toxin.
5. All listed.

4. What production processes may cause the formation of lead intoxication?
1. Battery assembly.
2. Installation of radio equipment.
3. Manufacturing and processing of products from crystal.
4. Pottery production using enamel and glaze.
5. All listed.

5. What type of poison is lead and its compounds?
1.The poison causing the formation of methemoglobin.
2.Yad blocking sulfhydryl groups of proteins.
3.Yad blocking respiratory enzymes in cells.
4. Poison blocking the blood coagulation system.
5.Yad activating the blood coagulation system (disseminated vascular coagulation syndrome - DIC).

6. What organs and tissue structures are affected by lead intoxication?
1. Blood system.

2.Central and peripheral nervous system.
3. Parenchymal organs (liver, kidneys).
4. Skin, mucous membranes, bones.
5. All listed.

7. In which organs and tissue structures is lead predominantly deposited during lead intoxication?
1. Liver
2. Kidneys.
3. Muscles.
4. Bones.
5. Deponded uniformly in all listed organs.

8. How is lead removed from the body?
1. Through the urinary system.
2. Through the biliary tract into the intestine.
3. Through sweat glands.
4. Through the mammary glands during lactation.
5. All listed.

9. What caused the severity of lead intoxication?
1. The amount of lead accumulated in the bones.
2. The amount of lead accumulated in the liver and kidneys.
3. Concentration of lead in circulating blood.
4. All of these circumstances.
5.None of the listed circumstances.

10. What pathological changes are not typical for lead toxicity?
1. Violation of the synthesis of porphyrins and heme.
2. The formation of methemoglobin in red blood cells.
3. Shortening the life of red blood cells (premature hemolysis).
4. Violation of the function of the peripheral centers of the vegetative nervous regulation with the appearance of spastic-atonic changes in the function of the intestine.
5. All are typical.

11. What syndromes is the clinical picture of chronic lead intoxication?
1. Syndrome of blood damage.
2. Syndrome lesions of the digestive system.
3. Syndrome of the nervous system.
4. Syndrome lesions skeler.
5. From all of the above.

12. Which of the following formulations are not part of the definition of the type of anemia that occurs during chronic lead intoxication?
1. Hypochromic anemia.
2. Sideroachrestic anemia.
3. Hypersiderinemic anemia.
4. Megaloblastic anemia.
5. Sideoblastic anemia.

1*3. What are the clinical and laboratory signs are not evidence of a violation of porphyrin metabolism in patients with chronic lead intoxication?*
1. Increase in the content of protoporphyrin in red blood cells.
2. Increase in urine coproporphyrin.
3. Increased urinary delta aminolevulinic acid.
4. Increased urine hemosiderin.
5. Urine dyeing with reddish porphyrin.

14. What pathological changes contribute to the grayish-earthy staining of the skin (lead color) of patients with chronic lead intoxication?
1. An increase in the content in the blood and in the skin of lead compounds.
2. An increase in the content in the blood and in the skin of porphyrins.
3. Anemia.
4. Spasm of blood vessels.
5. All listed.

15. What changes in the general analysis of blood are typical for chronic lead intoxication?
1. Reducing the number of red blood cells, color indicator is less than one.
2. Reducing the number of red blood cells, a color indicator greater than one.
3. Microsferocytosis.
4. Increasing the content of reticulocytes and young erythrocytes with basophilic granularity of cytoplasm.
5. The absence of reticulocytes and young erythrocytes with basophilic granularity of cytoplasm.

16. What signs of damage to the digestive organs are not typical for chronic lead intoxication?
1. Sensation of the presence of a foreign object ("hair") in the mouth.
2. Metallic taste of own saliva.
3. The lead border in the form of a bluish-black strip on the edge of the gingiva.
4. Suppression of the function of the salivary glands (xerostomia).
5. Cramping abdominal pain around the navel, unstable stool with irregularly alternating periods of diarrhea and constipation.

17. What are the typical symptoms of bouts of lead?
1.Intensive colicy pains in the abdomen, spreading around the navel and in the epigastric region.
2.Palpator study of the abdomen increases colic.
3. The abdominal wall becomes sharply strained, retracted.
4. Painful attacks accompanied by bloody diarrhea.
5.Palpation of the abdomen relieves pain.

1*8. What are the typical symptoms of bouts of lead?*
1.Can form a constipation that is not amenable to the action of weak agents.
2. Cal becomes fragmented, takes the form of sheep-it.
3. Blood pressure is increased.
4. The body temperature rises.
5. All are typical.

19. What syndrome that is formed during chronic lead intoxication is manifested by general weakness, fatigue, low emotional tone, memory deterioration, the formation of a neurocirculatory triad (hypothermia, bradycardia, arterial hypotension), the development of psychopathic disorders in severe cases ?

1. Astenovegetative syndrome.
2. Polyneuropathy syndrome.
3. Encephalopathy syndrome.
4. All listed.
5. None of the above.

20. What syndrome that is formed during chronic lead intoxication manifests itself as a painless peripheral neuritis, weakness of extensor muscles?

1. Astenovegetative syndrome.
2. Polyneuropathy syndrome.
3. Encephalopathy syndrome.
4. All listed.
5. None of the above.

21. What syndrome that is formed during chronic lead intoxication is manifested by impaired function of the cranial nerves (anisocaria, twitching of individual muscle groups, attacks, and dysarthria), convulsive seizures, cerebral vascular crises with hemiparesis, ophthalmoplegia?

1. Astenovegetative syndrome.
2. Polyneuropathy syndrome.
3. Encephalopathy syndrome.
4. All listed.
5. None of the above.

22. What radiological signs are typical for chronic lead intoxication?

1. Changes in the structure of the metaphysis of tubular bones in the form of dense transverse strips.
2. Usurations of small joints of hands.
3. Area of subchondral sclerosis, marginal osteophytes in the joints.
4. All are typical.
5. Everything is not typical.

23. In what form of chronic lead intoxication do asthenovegetative disorders predominate?

1. With a mild form.
2. With moderate form.
3. In severe form.
4. With all forms of clinical course of chronic saturism.
5. None of the mentioned forms of the clinical course of chronic saturnism.

24. In what form of chronic lead intoxication does the characteristic pale-earthy color of the skin appear, lead fringe on the gums, a moderately severe encepha-lopathy, a sensitive form of polyneuropathy, and pronounced asteno-vegetative disorders occur; characteristic is lead colic, hypochromic anemia, the appearance in the peripheral blood of reticulocytes and erythrocytes with basophilic granularity of cytoplasm?

1. With a mild form.
2. With moderate form.
3. In severe form.
4. With all forms of clinical course of chronic saturism.
5. None of the mentioned forms of the clinical course of chronic saturnism.

25. For what form of chronic lead intoxication is characteristic lead colic, severe lesions of the central and peripheral nervous system with sensory-motor disorders, preferential weakness of the extensors of the hands and fingers, pronounced hypochromic, sideroachresis, hyper-siderinemic, sideroblastic anemia ?
1. With a mild form.
2. With moderate form.
3. In severe form.
4. With all forms of clinical course of chronic saturism.
5. None of the mentioned forms of the clinical course of chronic saturnism.

26. What diagnostic criteria are typical for chronic lead intoxication?
1. Professional route - work in conditions of exceeding the maximum allowable concentrations of lead aerosols in the air, other circumstances suggesting the occurrence of acute or chronic intoxication with this metal in the workplace.
2. Asthenic syndrome, polyneuropathy, encephalopathy.
3. Lead coloring - earthy-pale color of skin coverings, caused by anemia, spasm of skin vessels, an increase in the content of protoporphyrin in erythrocytes.
4. The lead edge on the gums.
5. All are typical.

27. What diagnostic criteria are not typical for chronic lead intoxication?
1. Lead colic.
2.Reticulocytosis, erythrocytes with basophilic granularity in peripheral blood.
3. Hypochromic anemia.
4. The high content of hemosiderin in the urine.
5. Increased serum iron content.

28. What diagnostic criteria are not typical for chronic lead intoxication?
1. Increasing the number of sideroblasts in the bone marrow.
2. Increased color index of blood.
3. High content of protoporphyrin in red blood cells.
4. Increasing the concentration in the urine of coproporphyrin and delta-aminolevulinic acid.
5. Detection of lead in urine.

29. What drugs for oral administration are required if you need to remove lead ions from the victim's body?
1.Succimer.
2. Atropin.
3. Pipolphen.
4. D-penicylamine (cuprenyl).
5. All listed.

30. What medicines for parenteral administration should be prescribed, if necessary, removal of lead ions from the body of the victim?
1. Dimercaptol.
2. Thetacin-calcium.
3. Etilenediaminetetraacetate (EDTA) disodium salt.
4. Pentacin.
5. All listed.

31. What medicines should be prescribed for the purchase of lead colic?
1. Atropin.
2. Fentanyl.
3. Pipolfen, tavegil.
4. All listed.
5. None of the above.

32. What symptomatic drugs should be prescribed for the occurrence of polyneuropathy caused by lead intoxication?
1.Notropil.
2. Winpocetin.
3.Tocopherol.
4. Emoxipin.
5. All listed.

33. What are the ways tetraethyl lead can get into the human body?
1. Inhalation through the respiratory system.
2. Orally through the digestive tract.
3. Through intact skin in direct contact with poison.
4. Through the mucous membranes in the presence of poison on the conjunctiva of the eye.
5. All of the above.

34. What toxic effects are not typical for tetraethyl lead?
1. Neurotropic toxic effect.
2. Cytoplasmic toxic effect.
3. Capillary toxic effect.
4. Hemocoagulation effect.
5. Violation of porphyrin metabolism with the formation of sideroaesthetic anemia.

35. What stages are distinguished in the clinical picture of acute intoxication with tetraethyl lead?
1. The initial.
2. Pre-Culinary.
3. Culminating.
4. Predelirious.
5. All mentioned.

36. What syndromes form the clinical picture of the initial stage of acute intoxication with tetraethyl lead?
1. Hemolytic.
2. Anemic.

3. Asthenic.
4.Organic (pseudoparalitic encephalopathic).
5.Predelirious.

37. What is the initial stage of acute intoxication syndrome with tetraethyl lead characterized by severe headache, severe general weakness, fatigue, insomnia, emotional instability, hypersalivation, bradycardia, hypotension, lower body temperature, paresthesia?
1. Asthenic.
2.Organic.
3.Predelirious.
4. Any of the above.
5. None of the above.

38. What is the initial stage of acute intoxication syndrome with tetraethyl lead characterized by euphoria, resembling acute alcohol intoxication, instability of gait, nystagmus, dysarthria, trembling fingers, twitching hands, feet, torso?
1. Asthenic.
2.Organic (pseudoparalytic encephalopathic).
3. Predelirious.
4. Any of the above.
5. None of the above.

39. What is the initial stage of acute intoxication syndrome with tetraethyl lead characterized by hypersalivation, hyperhidrosis, sensation of the presence of hair in the mouth, crawling of insects on the body, a sense of fear of persecution, inevitable death, frightening gal-lucinations?
1. Asthenic.
2. Organic.
3. Predelirious.
4. Any of the above.
5. None of the above.

40. What stage of acute tetraethyl lead poisoning is characterized by pronounced vegetative disorders combined with general psychic arousal, delusional state, when frightening auditory, tactile, visual hallucinations appear against the background of darkened consciousness, the victims become aggressive, dangerous for others?
1. The initial.
2. Pre-Culinary.
3. Culminating.
4. Any of the above.
5. None of the above.

41. What stage of acute tetraethyl lead poisoning is characterized by abrupt psychomotor agitation with blackout, hallucinations, epileptiform convulsive seizures with subsequent transition to a comatose state with meningeal phenomena, heart failure, a vascular ring?
1. The initial.
2. Pre-Culinary.
3. Culminating.

4. Any of the above.
5.No for one of the listed.

42. What stage of chronic intoxication with tetraethyl-lead is characterized by a neurosis-like state, manifested by general weakness, fatigue, a combination of a depressive state with increased irritability, nightmarish dreams, an unreasonable feeling of fear, senestopathic sensations of an extraneous object (hair) in the mouth?
1.I stage.
2.II stage.
3.III stage.
4. Any of the above.
5. None of the above.

43. What stage of chronic intoxication with tetraethyl-lead is characterized by a progressive decrease in intelligence, memory, inadequate behavior, neurological disorders in the form of fingers trembling, impaired coordination of movements, instability of gait, dysarthria, nystagmus, the appearance of signs of sideroacresis anemia?
1.I stage.
2.II stage.
3.III stage.
4. For any of the listed stages.
5.No for one of the listed stages.

44. At what stage of chronic intoxication of tetraethyl-lead are profound mental status disorders with a predominance of psychomotor agitation, aggressiveness, extra-rapiramid and autonomic-sensory neuropathic disorders, severe sideroachrestric anemia?
1.I stage.
2.II stage.
3.III stage.
4. Any of the above.
5. None of the above.

45. What methods are not used in the treatment of acute intoxication with tetraethyl lead?
1. Wash the stomach with a suspension of activated carbon.
2. To give inside a fatty laxative (vaseline, castor oil).
3. Wash the poison from the skin with kerosene, then wash the skin with soapy water.
4. Introduce a parenteral solution of hexenal or barbamil.
5. Parenterally enter magnesia sulphate.

46. What drugs can not be used in the treatment of acute and chronic intoxication with tetraethyl lead?
1. Morphine.
2. Chloral hydrate.
3. Bromides.
4. All listed cannot be applied.
5. All listed can be applied.

47. What are the ways of penetration of poison into the body are typical for benzene intoxication?
1. Through the respiratory tract.
2. Through intact skin.
3. Through the mucous membranes.
4. Through the digestive tract.
5. All listed

48. What are the mechanisms of inactivation and removal of poison typical for intoxication with benzene and other aromatic hydrocarbons?
1. Compound in the liver with sulfuric and glucuronic acid with the formation of low-toxic substances.
2. The removal of poison partially unchanged, partly in the form of metabolites through the lungs.
3. The removal of poison partially unchanged, partly in the form of metabolites through the biliary system and intestines.
4. The removal of poison in the form of hydrophilic metabolites through the urinary-excretory system.
5. All are typical.

49. What pathological changes are possible with the elimination of ami-do- and nitro-derivatives of benzene through the urinary system?
1. Pyelonephritis.
2. Cystitis
3. Cancer of the bladder.
4. All listed.
5. None of the above.

50. What toxic effects are typical for acute poisoning with benzene and other aromatic hydrocarbons?
1. Narcotic lesion of the nervous system.
2. The defeat of the hematopoietic system.
3. Carcinogenic effect.
4. Decrease in the body's content of vitamins B6, B12 and C.
5. All are typical.

51. What toxic effects are typical for chronic poisoning with benzene and other aromatic hydrocarbons?
1. The defeat of the hematopoietic system.
2. Carcinogenic effect.
3. Myocardial dystrophy, fatty hepatosis.
4. All are typical.
5. Everything is not typical.

52. What syndrome arising during intoxication with aromatic hydrocarbons is characterized by emotional lability, loss of the ability to intensive mental labor, sleep disorders?
1. Asthenic syndrome.
2. Polyneuritic syndrome.

3. Toxic encephalopathy syndrome.
4. Syndrome of the funicular myelosis.
5. All listed.

53. What syndrome arising during intoxication with aromatic hydrocarbons is characterized by vegetative disorders, sensitivity disorders, decrease in skin temperature, excessive sweating, swelling of fingers, pulse mobility, blood pressure, pain, paresthesia, decrease in skin sensitivity ?
1. Asthenic syndrome.
2. Polyneuritic syndrome.
3. Toxic encephalopathy syndrome.
4. Syndrome of the funicular myelosis.
5. All listed.

54. What syndrome arising during intoxication with aromatic hydrocarbons is characterized by organic neurological symptoms with dyscirculatory disorders, extrapyramidal hyperkinesis, can manifest itself as psychotic deviations?
1. Asthenic syndrome.
2. Polyneuritic syndrome.
3. Toxic encephalopathy syndrome.
4. Syndrome of the funicular myelosis.
5. All listed.

55. What syndrome arising from intoxication with aromatic hydrocarbons is characterized by toxic damage to spinal cord structures resulting from deficiency of vitamin B12 and manifested by a decrease in deep muscle sensitivity, a decrease in Achilles reflexes, weakness in the legs, and a lack of coordination of movements?
1. Asthenic syndrome.
2. Polyneuritic syndrome.
3. Toxic encephalopathy syndrome.
4. Syndrome of the funicular myelosis.
5. All listed.

56. What hematological changes are typical for chronic intoxication with benzene and other aromatic hydrocarbons?
1. Granulocytopenia, agranulocytosis.
2. Thrombocytopenia.
3. Megaloblastic anemia.
4.Aplastic anemia.
5. All are typical.

57. What hematological changes are typical for chronic intoxication with benzene and other aromatic hydrocarbons?
1.Aplastic anemia.
2. Acute non-lymphoblastic leukemia.
3. Chronic myeloid leukemia.
4. All are typical.
5. Everything is not typical.

58. What is the severity of clinical manifestations of acute poisoning with benzene, similar to alcohol intoxication?
1. Mild intoxication.
2. Intoxication moderate.
3. Severe intoxication.
4. Any of the above.
5. None of the above.

59. What is the severity of clinical manifestations of acute benzene poisoning in the form of encephalopathy with loss of consciousness, muscle twitching, tonic and clonic seizures?
1. Mild intoxication.
2. Intoxication moderate.
3. Severe intoxication.
4. Any of the above.
5. None of the above.

60. What is the severity of clinical manifestations of acute poisoning with benzene in the form of almost instantaneous loss of consciousness followed by respiratory arrest and death?
1. Mild intoxication.
2. Intoxication moderate.
3. Severe intoxication.
4. Any of the above.
5. None of the above.

61. What is the severity of clinical manifestations of chronic intoxication with benzene in the form of a neurasthenic or asthenic syndrome with vegetative dysfunction, and also hematological changes in the form of leukopenia with neutropenia, relative lymphocytosis, moderately pronounced thrombocytopenic purpura?
1. Light degree.
2. The average degree.
3. Heavy degree.
4. Any of the above.
5. None of the above.

62. What is the severity of the clinical phenomenon of pro-chronic toxicity of benzene in the form of polyneuritis, symptoms funicular myelosis, encephalopathy combined with severe leucopenia, thrombocytopenia with common hemorrhagic purpura, anemia, caused by the defeat of the Erie tropoeticheskogo germ in the bone marrow, and bleeding?
1. Light degree.
2. The average degree.
3. Heavy degree.
4. Any of the above.
5. None of the above.

63. Which of the severity correspond to the clinical manifestations of chronic intoxication with benzene in the form of severe encephalopathy, agranulocytosis, deep thrombocytopenia, anemia, and severe hemorrhagic syndrome caused by the

devastation of the red bone marrow, in some cases in the marrow and peripheral blood of the large number of blast cells (acute leukemia)?
1. Light degree.
2. The average degree.
3. Heavy degree.
4. Any of the above.
5. None of the above.

64. Which of the following criteria are used for the diagnosis of intoxication with benzene and its compounds?
1. Professional route - proof of work in contact with aromatic hydrocarbons.
2. Identification of signs of damage to bone marrow hematopoiesis (agranulocytosis, thrombocytopenia, aplastic anemia).
3. The occurrence of acute "benzene" hemoblastosis or heme-tosarcoma.
4. Signs of toxic encephalopathy, damage to the liver, heart.
5. All listed.

65. Which of the following options for the penetration of mercury into the body is the least dangerous for humans?
1. Inhalation of metallic mercury vapors or aerosols of its compounds.
2. Oral ingestion of soluble mercury compounds into the digestive tract.
3. Oral ingress into the digestive tract of metallic mercury.
4. All listed.
5. None of the above.

*6***6. What organs and tissues can deposit mercury?**
1. Liver
2. Kidneys.
3. The brain (pituitary, cerebellum).
4. Bones of the skeleton.
5. All listed.

67. What ways of poison excretion are not typical for mercury intoxication?
1. Through the biliary system and the digestive tract.
2. Through the respiratory system.
3. Through the urinary system.
4. Through the salivary glands.
5. Milk glands during lactation.

68. What toxicological characteristics are typical for mercury intoxication?
1. Mercury - thiol poison, blocks sulfhydryl groups in protein compounds.
2. Mercury causes functional, and then degenerative changes in the central and peripheral nervous system.
3. Mercury causes a variety of pathological changes in the tissues of parenchymal organs.
4. All are typical.
5. Everything is not typical.

69. What symptoms are not typical for acute mercury intoxication?

1. Neurotoxicosis in the form of intense headache, nausea, vomiting, severe weakness, adynamia.
2.Metal taste in the mouth, salivation, abdominal pain, bloody diarrhea, polyuria.
3. Ulcerative stomatitis and gingivitis.
4. Thrombocytopenic purpura.
5. All are typical.

70. What is mercury eretism?
1. Unusual behavioral reaction in the form of extremely pronounced timidity, embarrassment, strong emotional excitement with a heartbeat, flushing of the face, sweating even in a familiar environment, and among familiar people.
2. Complex asymmetric extrapyramidal, large-scale intentional tremor against the background of small-amplitude asymmetric functional tremor.
3. Sensations of metallic taste in the mouth, gingivitis with a purple edge on the gums.
4. "Unreasonable" attacks of severe headaches.
5. Small-amplitude tremor of the fingertips.

71. Which severity corresponds to clinical manifestations of chronic mercury intoxication in the form of vascular dystonia with a neurosis-like syndrome in the form of pathological "embarrassment", emotional instability, vasomotor hyperreactivity, "causeless" attacks of strong head pains, sensations of metallic taste in the mouth , mercury gingivitis with purple rims on the gums, gastric dyspepsia, unstable blood pressure, tachycardia, small amplitude tremor of the fingertips?
1. Light degree.
2. The average degree.
3. Heavy degree.
4. Any of the above.
5. None of the above.

72. Which severity corresponds to the clinical manifestations of chronic mercury intoxication in the form of organic disruptions in the central nervous system with pronounced manifestations of mercury eretism, complex asymmetric extrapyramidal, large-scale intentional tremor (flapping) against a small amplitude asymmetric function; pa, a sensitive form of polyneuropathy, gingivitis with an intense lilac border on the gums, stomatitis, signs of toxic hepatitis, myocardial dystrophy?
1. Light degree.
2. The average degree.
3. Heavy degree.
4. Any of the above.
5. None of the above.

73. What is the severity of clinical manifestations of chronic mercury intoxication in the form of toxic encephalopathy, persistent organic changes in the nervous system, mercury eretism, reaching extreme severity, extra-pyramidal large-scale tremor, taking a generalized character?
1. Light degree.
2. The average degree.
3. Heavy degree.
4. Any of the above.

5. None of the above.

74. What criteria can not be used to confirm the diagnosis of professionally caused mercury intoxication?
1. Professional route, certifying long-term contact in the production environment with mercury and its compounds.
2. A violet border on gums, gingivitis, stomatitis.
3. Clinical manifestations of mercury erethism, functional or extrapyramidal tremor (flapping).
4. Detection of free hemoglobin in the blood.
5. Identification of elevated levels of mercury in the blood, in the urine.

75. What medicines are used for the etiotropic treatment of mercury intoxication?
1.Desferal.
2.Succimer.
3. Unitiol.
4.Penitsilamin.
5. All listed.

76. What pathological process is the key to poisoning organophosphate pesticides?
1. Blockade of sulfhydryl groups in protein compounds.
2. Blockade of phosphodiesterase.
3. The formation of methemoglobin.
4. Hemolysis.
5. Activation of plasma coagulation factors.

77. What pathological processes are typical for organophosphate pesticide intoxication?
1. As a result of the blockade of phosphodiesterase, large amounts of acetylcholine accumulate in synapses.
2. M-cholinergic reactions arise in the form of constriction of the pupils, bronchospasm, bronchial hypersecretion, pathological activation of the secretory and motor function of the stomach.
3. H-cholinergic reactions appear in the form of fibrillar muscle twitches, mental arousal.
4. All are typical.
5. Everything is not typical.

78. Which severity corresponds to phosphorus-organic pesticides poisoning, if the victims are worried about intense sweating, drooling, abdominal pain, nausea, vomiting with abundant acidic liquid contents, expiratory dyspnea, a cough with a large amount of bright sputum, they become agitated and, together so that they are adynamic, their pupils sharply narrow, the arterial pressure rises, the pulse increases?
1. Easy poisoning.
2. Medium poisoning.
3. Severe poisoning.
4. The degree of severity of intoxication for these symptoms cannot be determined.
5. Clinical manifestations are not typical for intoxication with phospho-ororganic pesticides.

79. What kind of severity corresponds to phosphorus-organic pesticides poisoning, if the affected skin becomes pale marble, fibrillation of the tongue, eyelids, other muscle groups, involuntary jerking movements of the eye-apples, difficulty in speech, mental disorders with severe depression occur , hallucinations, twilight state, fever up to 400C possible?

1. Easy poisoning.
2. Medium poisoning.
3. Severe poisoning.
4. The degree of severity of intoxication for these symptoms cannot be determined.
5. Clinical manifestations are not typical for intoxication with phospho-ororganic pesticides.

80. What stages of severe phosphorus-organic pesticide poisoning correspond to clinical manifestations in the form of abundant sweat, salivation, tearing, bronchorea, difficulty breathing, visual impairment, and abdominal pain on the background of psychic arousal?

1. Stage of arousal.
2. The convoy stage.
3. The paralytic stage.
4. The stage of severe poisoning with the indicated symptoms cannot be determined.
5. Clinical manifestations are not typical for intoxication with phospho-ororganic pesticides.

81. Which stages of severe poisoning with organophosphate pesticides correspond to clinical manifestations in the form of adynamia of the twilight state of the psyche, clonic-tonic muscle cramps, hypoxemia caused by a severe asthmatic condition, symptoms of toxic damage to the liver, kidneys, heart?

1. Stage of arousal.
2. The convoy stage.
3. The paralytic stage.
4. The stage of severe poisoning with the indicated symptoms cannot be determined.
5. Clinical manifestations are not typical for intoxication with phospho-ororganic pesticides.

82. What stage of severe poisoning with phosphorus-organic pesticides corresponds to clinical manifestations in the form of a comatose state, acidosis, with a high probability of death as a result of pulmonary edema, cardiovascular insufficiency, respiratory muscle pair?

1. Stage of arousal.
2. The convoy stage.
3. The paralytic stage.
4. The stage of severe poisoning with the indicated symptoms cannot be determined.
5. Clinical manifestations are not typical for intoxication with phospho-ororganic pesticides.

83. What symptoms are not typical for the initial period of chronic intoxication with organophosphate pesticides?

1. General weakness, loss of mental and physical ability, memory impairment, headache, dizziness.
2. Red, persistent dermographism.
3. Petechial hemorrhagic rash.
4. Blood pressure decreases.
5. The pulse rate decreases.

84. What pathological manifestations are not typical for long-term chronic toxicity with organophosphate pesticides?
1. Painful headaches.
2. Dreams with frightening nightmares.
3. Muscle twitching, paresthesia.
4. Spastic paralysis of various muscle groups.
5. Cold urticaria.

85. Which of the following biochemical **changes are typical for intoxication with organophosphate pesticides?**
1. Increase the level of bilirubin in the blood.
2. Decrease in the activity of pseudo-cholinesterase in the blood.
3. Reducing the content of prothrombin in the blood.
4. Increased alkaline phosphatase activity.
5. All listed.

86. Which of the following should be used to confirm the diagnosis of professionally caused toxic organophosphate pesticides intoxication?
1. Professional route, confirming the probability of contact of the victim with organophosphate toxic chemicals under production conditions.
2. The results of the hygienic examination of the workplace of the victim, confirming the possibility of occupational toxicity.
3. A characteristic clinical picture of acute or chronic organophosphate toxic chemicals.
4. Identification of a low level of activity of pseudo-coline estes in the blood.
5. All of the above.

87. What symptoms do not allow differentiating intoxication with organophosphate pesticides from poisoning with other poisons used in agriculture?
1. Drastic constriction of the pupils.
2. Bradycardia.
3. Headache.
4. Pronounced activation of the secretory function of salivary, lacrimal, gastric, bronchial glands.
5. Reducing the activity of pseudo-cholinesterase in the blood.

88. What antidotes of pathogenetic action are used to relieve acute intoxication with organophosphate pesticides if M-cholinergic reactions prevail?
1. Atropin.
2. Alloxime.
3. Izonitrosin or dipyroxime.
4. Benzogeksony or pentamine.
5. All listed.

89. What antidotes of pathogenetic action are used to relieve acute intoxication with organophosphate pesticides, if H-cholinergic reactions prevail?
1. Atropin.
2. Benzohexony or pentamine
3. Alloxime.
4. Izonitrosin or dipyroxime.
5. All listed.

90. What is the characteristic of organochlorine pesticides that limits their use in agriculture?
1. High toxicity.
2. The difficulties of industrial production and transportation.
3. High resistance to degradation in natural conditions.
4. All the features mentioned.
5.None of the features mentioned.

91. What are the peculiarities of toxic action are not typical for organochlorine pesticides poisoning?
1. A generally toxic effect on the human body.
2. The ability to penetrate the lipid layer of the cell membrane and inhibit the intracellular enzymes of the respiratory cycle.
3. Ability to form methemoglobin.
4. The ability of certain substances of this group to block the thiol groups of protein compounds.
5. First of all, the brain (mainly subcortical region) and the liver are affected.

92. What are the typical symptoms of acute chlorine pesticide poisoning?
1. A sharp weakness, nausea, vomiting, intense headache, dizziness, fever up to 39-400C.
2. General inhibition, mental disorders, trembling, in jerking of various muscle groups, attacks of clonic-tonic seizures.
3. Frequent, noisy breathing (decompensated metabolic acidosis).
4. Anuria, jaundice.
5. All are typical.

93. Which syndromes do not participate in the formation of the clinical picture of chronic toxicity with organochlorine pesticides?
1. Astenovegetative syndrome.
2. Polyneuritic syndrome.
3.Cardial syndrome.
4. Hepatic syndrome.
5. Thromboembolic syndrome.

94. What is the syndrome that occurs during chronic intoxication with organochlorine pesticides characterized by progressive general weakness, reduced physical and, especially, mental disability, headaches, dizziness, sweating, emotional lability, palpitations, interruptions in heart rhythm?
1. Polyneuritic syndrome.
2.Cardial syndrome.

3. Hepatic syndrome.
4. Astenovegetative syndrome.
5. The indicated symptoms are not typical for intoxication with phosphorus pesticides.

95. What is the syndrome that arises during chronic intoxication with organochlorine pesticides characterized by pain along the nerve trunks, trembling, muscle twitching, impaired skin sensitivity, visual disturbances?
1. Polyneuritic syndrome.
2.Cardial syndrome.
3. Hepatic syndrome.
4. Astenovegetative syndrome.
5. The indicated symptoms are not typical for intoxication with phosphorus pesticides.

96. For what syndrome that arises during chronic intoxication with organochlorine pesticides, there is a tendency to lower blood pressure, rapid pulse, supraventricular and ventricular extrasystole, blockade of the cardiac conduction system, signs of myocardial dystrophy are recorded (according to ECG and EchoCG)?
1. Polyneuritic syndrome.
2. Hepatic syndrome.
3. Astenovegetative syndrome.
4.Cardial syndrome.
5. The indicated symptoms are not typical for intoxication with phosphorus pesticides.

97. For which syndrome arising from chronic organochlorine intoxicating pesticides are episodes of hypoglycemic states, ikterichnost sclera, skin, moderate hepatomegaly, hyperbilirubinemia, increased activity of AST, AST, LDH, with ultrasound signs of steatohepatitis (increased volume liver, diffuse increase in echogenicity of the parenchyma, moderately pronounced disorders of portal hemodynamics)?
1. Polyneuritic syndrome.
2. Hepatic syndrome.
3. Astenovegetative syndrome.
4.Cardial syndrome.
5. The indicated symptoms are not typical for intoxication with phosphorus pesticides.

98. Which of the following circumstances serve as proof of professionally caused chlorine-pesticide toxicity intoxication?
1. Professional route confirming the likelihood of contact of the victim with organochlorine toxic chemicals under production conditions.
2. The results of the hygienic examination of the workplace of the victim, confirming the possibility of professional toxicity with organochlorine pesticides.
3. A characteristic clinical picture of acute or chronic organochlorine toxic chemicals.
4. All listed.
5. None of the above.

99. Which of the following should be used in the provision of emergency care in case of acute poisoning with organochlorine pesticides?
1. To establish inhalation of moistened oxygen.
2. When psychomotor agitation, convulsive syndrome, intravenous hexenal is administered.
3. In case of acidosis, inject 200-400 ml of 2% sodium bicarbonate solution intravenously.
4. Parenterally administer glucose solutions, vitamins C, B1, cocarboxylase, pyridoxine, calcium gluconate.
5. All of the above.

100. What medicines are not indicated for the treatment of chronic toxicity with organochlorine pesticides?
1. Course detoxification therapy with a solution of 5% glucose, saline solutions, reopolyglucine.
2. Daily oral intake of balanced multivitamin preparations.
3.Hepatoprotectors - Essentiale-Forte, lipostabil, lipamid.
4.Blockers of beta-adrenergic receptors.
5. All are shown.

101. What toxic effects are typical for organo-mercury pesticides-fungicides?
1. Blockade of sulfhydryl groups of proteins.
2. Capillarytoxic effect.
3. Cardiotoxic action.
4. The defeat of the genital glands and ovaries in women.
5. All are typical.

102. What symptoms are not characteristic of mild and moderate cravings of acute toxicity of organo-mercury organic fungicide pesticides?
1. Metallic taste, burning sensation in the mouth.
2. Progressive general weakness, headache, dizziness, nausea, vomiting.
3. Severe thirst, intense abdominal pain, diarrhea, often bloody.
4. Arterial hypotension.
5. Loss of consciousness with involuntary urination and deflation.

103. What symptoms are not typical for severe acute organ mercury toxicity intoxication?
1. Unstable gait, trembling hands, trunk.
2. Difficult swallowing.
3. Impaired vision until blindness.
4. Loss of consciousness with involuntary urination and defecation.
5. All are characteristic.

104. To which syndrome, which is included in the clinical picture of chronic intoxication of organo-mercury pesticides-fungicides, do complaints of headache, dizziness, reduced physical and mental disability, impaired memory, increased emotional lability correspond?
1. Astenovegetative syndrome.
2. Polyneuritic syndrome.
3. Diencephalic-hypothalamic syndrome.

4.Cardial syndrome.
5. Hepatic syndrome.

105. For what syndrome arising during chronic intoxication of organo-mercury pesticides-fungicides, is there an increase in tendon reflexes, trembling of fingers, formation of encephalomyeloradiculoneuritis with focal and diffuse manifestations, epileptiform convulsive attacks?
1. Astenovegetative syndrome.
2. Diencephalic-hypothalamic syndrome.
3. Polyneuritic syndrome.
4.Cardial syndrome.
5. Hepatic syndrome.

106. What is the syndrome that occurs during chronic intoxication of organo-mercury pesticides-fungicides, characterized by impaired thermoregulation, increased thirst, polyuria, insomnia, frightening nightmarish dreams with battle scenes, psychotic crises with feelings of causeless anguish, fear?
1. Astenovegetative syndrome.
2. Polyneuritic syndrome.
3. Diencephalic-hypothalamic syndrome.
4.Cardial syndrome.
5. Hepatic syndrome.

107. For what syndrome arising from chronic intoxication of organo-mercury pesticides-fungicides, are arterial hypotension, bradycardia, supraventricular and / or ventricular premature beats, myocardiodystrophy with signs of circulatory failure?
1. Astenovegetative syndrome.
2. Polyneuritic syndrome.
3. Diencephalic-hypothalamic syndrome.
4.Cardial syndrome.
5. Hepatic syndrome.

108. For what kind of syndrome that occurs during chronic intoxication of organo-mercury pesticides-fungicides, moderate hepatomegaly, impaired protein-forming liver function, slight hyperbilirubinemia, increased AST, ALT, LDH, gamma-glutamyl transpeptidase, detection of UZI are characteristic; (increase in liver mass, diffusive increase in echogenicity, depletion of vascular parenchyma pattern)?
1. Astenovegetative syndrome.
2. Polyneuritic syndrome.
3. Diencephalic-hypothalamic syndrome.
4.Cardial syndrome.
5. Hepatic syndrome.

109. Which of the following criteria should be used to confirm the diagnosis of a professionally determined toxicity of organo-mercury pesticides-fungicides?
1. Professional route, confirming the probability of contact of the victim with organic mercury pesticides in the workplace.
2. The results of the hygienic examination of the workplace of the victim, confirming the possibility of professional toxicity to organo-mercury pesticides.

3. A characteristic clinical picture of acute or chronic organ mercury toxicity poisoning.
4. Detection of mercury in the urine of the victim.
5. All listed.

110. Which of the following methods should not be used in the provision of emergency assistance in case of poisoning with mercury-organic pesticides-fungicides?
1. Remove the victim from the zone contaminated with pesticides.
2. Remove contaminated clothing, wash the skin with warm water, alcohol-soda solution.
3. When the chemical enters the digestive tract, wash the stomach with two or three liters of water containing 100 ml of Strizhevsky anti-dota.
4. Conduct antidote therapy.
5. Give a laxative (castor or linseed oil).

111. What medicines are used as antidotes in the treatment of intoxication with mercury-containing fungicide pesticides?
1. Unitiol.
2. Pentacin.
3.Desferal.
4. Atropin.
5. All listed.

112. What medicines should be used to treat chronic intoxication with mercury-containing fungicide pesticides?
1. Unitiol.
2.Succimer.
3.Calcium gluconate, balanced multivitamin preparations.
4. Essentiale-forte, lipamid, lipostabil.
5. All listed.

113. What substances do not have an irritating effect on the respiratory organs?
1. Gas chlorine.
2. Carbon monoxide.
3. Ammonia.
4. Sulfur oxides.
5. Hydrogen chloride.

114. What pathological processes occur in the lungs when inhaled gaseous substances with irritant action and well soluble in water?
1. Irritation and cauterization of the mucous membrane of the upper respiratory tract.
2. Tracheobronchitis.
3. Toxic alveolitis.
4. Pneumonitis.
5. Acute toxic pulmonary edema.

115. What pathological processes occur in the lungs when inhaled gaseous substances with irritant effects, but relatively poorly soluble in water?

1. Irritation and cauterization of the mucous membrane of the upper respiratory tract.
2. Tracheobronchitis.
3. Toxic alveolitis.
4. Pneumonitis.
5. Acute toxic pulmonary edema.

116. What forms of pulmonary lesions are typical for acute respiratory depression of a toxic-chemical etiology?
1. Acute tracheobronchitis.
2. Acute toxic-chemical pulmonary edema.
3. Alveolith.
4. Pneumonitis.
5. Chronic bronchitis, pneumosclerosis.

117. What forms of lung lesions are typical for chronic lesions of the respiratory organs of a toxic-chemical etiology?
1. Acute tracheobronchitis.
2. Acute toxic-chemical pulmonary edema.
3. Alveolith.
4. Pneumonitis.
5. Chronic bronchitis, pneumosclerosis.

118. What features are not typical for the primary reaction in toxic-chemical lesion of the respiratory organs?
1. After inhalation of the irritating gas, there is an acute suffocating laryngospasm, bronchospasm.
2. The primary reaction is especially pronounced when inhaling water-soluble gases (chlorine, hydrogen chloride, ammonia).
3. The primary reaction is less bright when relatively small gases are soluble in water (oxides of azo) in the respiratory tract.
4. All are typical.
5. Everything is not typical.

119. What features are not typical for the latent period in toxic-chemical damage to the respiratory organs?
1. It appears immediately after the initial reaction and can last from 1 hour to 2 days.
2. During this period, the victim recovers good health, he seems fully recovered.
3. The hidden period has a longer duration when breathing in gases that are poorly soluble in water.
4. All are typical.
5. Everything is not typical.

120. What features are not typical for the period of developed clinical reactions in toxic-chemical lesion of the respiratory organs?
1. It begins suddenly, more often at night, with acute toxic-chemical pulmonary edema with the defeat of slightly irritating gases in water.
2. It begins suddenly, more often at night, with acute toxic-chemical tracheobronchitis with lesions that are well soluble in water in irritating gases.

3. Acute toxic-chemical pulmonary edema can occur in two versions - blue or gray.
4. All are typical.
5. Everything is not typical.

121. What shifts are characteristic of the blue variant of toxic-chemical pulmonary edema in case of lesion of the respiratory organs with irritant gases?
1. Severe intraalveolar exudation combined with the obstruction of the small bronchi.
2. The swelling of interstitial tissue, the combination of acute hypoxia with hypocapnia.
3. Ears with difficulty breathing in and out, coughing with frothy sputum.
4. Small circle hypertension, acute overload of the right heart.
5. All are characteristic.

122. What shifts are characteristic of the gray variant of toxic-chemical pulmonary edema in case of lesion of the respiratory organs with irritant gases?
1. Severe intraalveolar exudation combined with the obstruction of the small bronchi.
2. The swelling of interstitial tissue, the combination of acute hypoxia with hypocapnia.
3. Ears with difficulty breathing in and out, coughing with frothy sputum.
4. Small circle hypertension, acute overload of the right heart.
5. All are characteristic.

123. What clinical forms can acute toxic-chemical tracheobronchitis acquire during the period of developed clinical reactions?
1. Toxic chemical rhinitis.
2. Toxico-chemical pharyngolaryngotracheitis.
3. Toxico-chemical endobronchitis.
4. Toxic and chemical pulmonary edema.
5. All listed.

124. What clinical forms has a period of outcomes of toxic-chemical lesion of the respiratory organs?
1. Alveolitis with a flow duration of 2-3 weeks.
2. Pneumonitis with a duration of 2-3 weeks.
3.Infectious-inflammatory processes with a probable transition to a chronic form of pulmonary pathology.
4. All listed forms.
5.None of the listed forms.

125. What clinical forms are typical for chronic toxic-chemical damage to the respiratory organs?
1. Chronic obstructive bronchitis.
2. Bronchial asthma.
3. Diffuse pneumosclerosis, emphysema.
4. Chronic pulmonary heart.
5. All are typical.

126. Based on what criteria is the diagnosis of a professionally determined toxic-chemical lesion of the lungs confirmed?
1. Professional route showing the possibility of a chronic or acute (emergency) toxic-chemical damage to the respiratory organs.
2. The results of the hygienic assessment of the working conditions of the victim, with the identification of the gaseous substance that caused the professional disease.
3. A characteristic clinical picture typical of an acute or chronic toxic-chemical lesion of the bronchopulmonary system.
4. Results of laboratory and instrumental diagnostics, which gives an objective assessment of the severity of respiratory injury, the state of compensation of the pulmonary-cardiac system.
5. On the basis of all the listed criteria.

127. What emergency measures should be carried out in the acute period of toxic-chemical damage to the respiratory organs?
1. Inhalation of 1-2% sodium bicarbonate solution is performed.
2. Intramuscularly inject antihistamine and analgesic drugs.
3. In severe lesions, hydrocortisone is administered parenterally (25 mg intramuscularly after 6-8 hours).
4. In order to reduce vascular permeability, they give calcium gluconate (0.5 orally ingested 3 times a day).
5. All listed.

128. What measures need to be performed in the latent period of toxic-chemical damage to the respiratory organs?
1. To observe the victim, it is possible in the outpatient setting.
2. Assign oral administration of expectorant drugs.
3. Place the victim in the hospital.
4. Urgently perform an ECG, chest X-ray, complete blood count.
5. Subcutaneously enter with prophylactic promedol (0.5 ml of 2% solution) and atropine (0.5 ml of 0.1% solution).

129. What measures should not be performed in the developed clinical stage of toxic-chemical lesion of the respiratory organs in case of toxic pulmonary edema?
1. Oxygen in the form of 48% air mixture, previously saturated with vapors of ethyl alcohol, inhalation of antifoamilan antifoam (2-3 ml of 10% alcohol solution) through a nasal catheter.
2. Morphine - 1 ml of 1% solution or promedol - 1 ml of 2% solution intravenously slowly; 0.5 ml of a 0.1% solution of atropine subcutaneously.
3. Furosemide - 40 mg intravenously.
4. When the arterial pressure drops, dopamine is injected intravenously (250 mg is diluted in 200 ml of reopolyglucin), methylprednisolone - 90-120 mg intravenously.
5. Everything should be done.

130. What remedies are not used in the treatment of chronic respiratory diseases of toxic-chemical etiology?
1. Inhalation of sodium bicarbonate solution.
2.Antibiotics in the occurrence and exacerbation of secondary infectious-inflammatory processes in the bronchopulmonary system.

3. Veroshpiron.
4. Therapeutic exercise, massage of the chest.
5. All are applied.

131. What features are typical for carbon monoxide (carbon monoxide)?
1. It is formed as a result of incomplete oxidation of carbon and carbon-hydrogens under conditions of lack of oxygen.
2. Drops into the body through the respiratory tract.
3. Drops into the body through intact skin and mucous membranes.
4. Drops into the body through the digestive tract.
5. It is excreted unchanged through the urinary system.

132. What features are not typical for carbon monoxide intoxication?
1. Carbon monoxide, which entered the blood through the alveolar membrane, competes with oxygen for hemoglobin.
2. Carbon monoxide has a greater ability than oxygen to combine with hemoglobin.
3. Carbon monoxide poisoning can occur at significantly lower concentrations than oxygen in ambient air.
4. The carbon dioxide forms a stronger, compared with oxygen, compound with hemoglobin, called carboxyhemoglobin.
5. Carboxic hemoglobin has a toxic effect on the intracellular enzymes of the respiratory cycle.

133. What features are not typical for carbon monoxide intoxication?
1. When a large amount of carboxyhemoglobin is accumulated in the blood, it becomes unable to perform the function of transporting oxygen from the lungs to organs and tissues at normal atmospheric pressure.
2. The presence of carboxyhemoglobin in the blood causes hemic hypoxia of the internal organs.
3. In connection with the blockade of carbon monoxide by intracellular cytochromoxidase, tissue hypoxia occurs.
4. All are typical.
5. Everything is not typical.

134. What features are not typical for carbon monoxide intoxication?
1. The decay of carboxyhemoglobin accelerates with an increase in the content of carbon dioxide (carbon dioxide) in the blood.
2. The decay of carboxyhemoglobin is accelerated under the influence of the ultraviolet part of the spectrum of sunlight.
3.When chronic carbon monoxide intoxication increases the content of non-heme iron in the blood.
4. Negeminovoy iron in the blood can bind carbon monoxide.
5. All are typical.

135. What is the severity of clinical manifestations of acute carbon monoxide poisoning in the form of general weakness of shortness of breath, pulsating headache, dizziness, nausea, often vomiting, increased heart rate while maintaining arterial pressure within normal limits?
1. Easy degree of carbon monoxide poisoning.

2. Medium carbon monoxide poisoning.
3. Severe severity of carbon monoxide poisoning.
4. Residual effects of acute carbon monoxide intoxication.
5. The indicated clinical manifestations are not typical for carbon monoxide poisoning.

136. What is the severity of clinical manifestations of acute carbon monoxide poisoning in the form of general inhibition, drowsiness, frequent, shallow breathing, vomiting, a sharp increase in pulse rate, lower blood pressure, short-term loss of consciousness?
1. Easy degree of carbon monoxide poisoning.
2. Medium carbon monoxide poisoning.
3. Severe severity of carbon monoxide poisoning.
4. Residual effects of acute carbon monoxide intoxication.
5. The indicated clinical manifestations are not typical for carbon monoxide poisoning.

137. What is the severity of clinical manifestations of acute carbon monoxide poisoning in the form of toxic, hypoxic coma, tonic and clonic convulsions on the background of deceptive rigidity?
1. Easy degree of carbon monoxide poisoning.
2. Medium carbon monoxide poisoning.
3. Severe severity of carbon monoxide poisoning.
4. Residual effects of acute carbon monoxide intoxication.
5. The indicated clinical manifestations are not typical for carbon monoxide poisoning.

138. What pathological changes are not typical for acute carbon monoxide toxicity?
1. Toxic pulmonary edema.
2. Bright pink infiltrates on the skin, bullous necrotic changes of the dermis.
3. Zheltukha.
4. The presence of carboxyhemoglobin in the blood.
5. All are typical.

139. What residual changes are left behind by acute carbon monoxide poisoning?
1. Peripheral polyneuropathy.
2.Motor polyneuritis.
3. Toxic encephalopathy with severe psychasthenia, prone to psychosis.
4. Cirrhosis of the liver.
5. Chronic renal failure.

140. What are the typical shifts for chronic carbon monoxide toxicity?
1. Psychological disorders, vegetative disorders, angio dystonia.
2. Diencephalic crises.
3. Myocardial dystrophy.
4. All are typical.
5. Everything is not typical.

141. What changes in laboratory parameters are not typical for chronic carbon monoxide intoxication?
1. Moderate erythrocytosis, elevated hemoglobin content.

2. Increased serum iron content.
3. The increased content of protoporphyrin in red blood cells.
4. The excretion of coproporphyrin and delta-aminolevulinic acid with urine is increased.
5. All are typical.

142. What criteria should be used to confirm the diagnosis of a professionally caused carbon monoxide intoxication?
1. Reliable data on the presence of high carbon monoxide content in the working area.
2. Clinical signs characteristic of carbon monoxide intoxication.
3. Detection of a high content of carboxyhemoglobin in the victim's blood.
4. High blood levels of non-heme iron, delta-aminolevulinic acid and coproporphyrin in the urine during chronic toxicity with carbon monoxide.
5. The aggregate of all the mentioned criteria.

143. What methods are used in assisting a victim of carbon monoxide poisoning?
1. The person is taken out to the fresh air, warm.
2. Oxygen therapy in the chamber for hyperbaric oxygenation.
3.Barbamil 3 ml of 10% solution together with 1 ml of 1% solution of dimed-rola is injected intravenously with psychomotor agitation, convulsions.
4. All listed.
5. None of the listed methods is effective for acute carbon monoxide intoxication.

144. What medicines are not able to reduce the toxic effect of carbon monoxide?
1.Ferkaven.
2. Methylene blue.
3. Cococarboxylase.
4. Tsitokhrom S.
5. D-penicylamine.

ANSWERS TO CONTROL TESTS

Answers to test tests to section I

01 – 1
02 – 3
03 – 2
04 –3, 4
05 – 4
06 – 3, 4
07 – 1, 2, 3
08 – 1
09 – 2
10 – 3
11 – 4
12 – 1
13 – 2
14 – 3
15 – 4
16 – 4
17 – 1, 2
18 – 3, 4
19 – 1, 2, 3, 4, 5
20 – 3, 5
21 – 1
22 – 1, 2, 3, 4, 5
23 – 1, 2, 3, 4
24 – 2, 5
25 – 1, 2, 3, 4, 5
26 – 1, 4, 5
27 – 1, 2, 3
28 – 1, 4
29 – 1, 2, 4
30 – 1, 2
31 – 3, 4
32 – 5
33 – 1, 2, 5
34 – 1, 3, 4
35– 1
36 – 2
37 – 3
38 – 4
39 - 5
40 – 4, 5
41 – 2, 3
42 – 1
43 – 2, 3, 4, 5
44 – 1, 2, 3, 4
45 – 2
46 – 1, 2
47 – 3
48 – 1, 2, 3, 4
49 – 1, 3, 5
50 – 1, 2, 3, 5

Answers to test tests to section II

01 – 4
02 – 1
03 – 2
04 – 3
05 – 5
06 – 1
07 – 2
08 – 5
09 – 3
10 – 2
11 – 1
12 – 2
13 – 3
14 – 2
15 – 4
16 – 4
17 – 1
18 – 2
19 – 3
20 – 4
21 – 5
22 – 1
23 – 2
24 – 3
25 – 5
26 – 3
27 – 4
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35 – 4
36 – 4
37 – 1
38 – 3
39 – 3
40 – 3
41 – 4
42 – 4
43-- 4
44 – 3
45 – 4
46 – 3
47 – 4
48 – 3
49 – 2
50 – 3
51 – 4
52 – 4
53 – 5
54 – 5
55– 1, 2
56 – 1, 4
57 – 2
58 – 3, 5
59 – 1
60 – 1, 2
61 – 4
62 – 1, 2, 4
63 –- 3, 5

Answers to test tests to section III

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02 – 4
03 – 1
04 – 1, 2, 3
05 – 2, 3, 4
06 – 1
07 – 3, 4
08 – 3, 4
09 – 1
10 – 3
11 – 5
12 – 2
13 – 4
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20 – 5
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22 – 5
23 – 5
24 – 4
25 – 3
26 – 3
27 – 1
28 – 2
29 – 3
30 – 4
31 – 5

Answers to test tests to section IV

01 – 1, 2
02 – 5
03 – 1, 2
04 – 4
05 – 2, 3
06 – 2
07 – 1
08 – 5
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10 – 2, 3
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12 – 5
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31 – 5
32 – 1, 2, 5
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37 – 4
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40 – 1
41 – 4
42 – 3
43 – 5
44 – 1
45 – 2
46 – 3
47–4

Answers to test tests to section V

01 – 1, 4
02 – 1, 3
03 – 5
04 – 5
05 – 2
06 – 5
07 – 4
08 – 5
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103 – 5
104 – 1
105 – 3
106– 3
107 – 4
108 – 5
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112 – 5
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115 – 3, 4, 5
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117 – 3, 4, 5
118 – 4
119 – 4
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122 – 2, 4
123 – 1, 2, 3
124 – 4
125 – 5
126 – 5
127 – 5
128 – 3, 4, 5
129 – 5
130 – 5
131 – 1,2
132 – 5
133 – 4
134 – 5
135 – 1
136 – 2
137 – 3
138 – 3

139 – 1, 2, 3
140 – 4
141 – 5
142 – 5
143 – 4
144-5

EXPLANATION TEXTS
to test tests section I
"GENERAL QUESTIONS OF PROFESSIONAL PATHOLOGY"

Test 1. **Occupational diseases are damage to health caused by exposure to harmful and / or dangerous factors of the labor process. The level of risk of occupational pathology depends on the hygienic conditions at the workplace, the intensity and severity of the labor process, the presence of harmful factors potentially harmful to the health of workers.**

Test 2. **Professional pathology as a clinical discipline has deep historical roots. In the 16th century, for the first time, a number of diseases among miners resulting from inhaling the dust of rocks were described by miners. Over the next 300 years, in the countries of Europe, as a result of careful observations and detailed scientific research, quite complete ideas about occupational diseases and the causes causing them have been developed, which have made it possible to develop effective ways to prevent harmful effects in the workplace. In the Republic of Belarus, the service of occupational pathology has existed since 1925, when the Department of Occupational Pathology was established in the 1st clinical hospital in Minsk, which included 16 inpatient beds, clinical and experimental laboratories.**

Test 3. **In the XVI century, Agricola in his work "On Mining and Metallurgy" for the first time described a number of diseases in miners resulting from the inhalation of dust from rocks. In the same century, Paracelsus published clinical descriptions of a number of occupational diseases of miners and metal lurgov, for the first time pointed out the true causes of their occurrence. In 1700, Bernardo Romazzini wrote the first book devoted to the issues of occupational pathology - "On the diseases of artisans".**

Test 4. **In the *Republic of Belarus there is a service of professional pathology*. Its main tasks are:**
• development and implementation of measures to develop and improve the service of occupational pathology;
• provision of specialized medical care to the population of the Republic of Belarus.
The purpose of the work of the service of professional pathology is professional selection, health promotion of workers, prevention, early diagnosis, treatment and medical rehabilitation of occupational diseases.
The structure of the service of occupational pathology consists of three levels: republican, regional and city (district).
Republican level of service of professional pathology:
• Republican Center for Occupational Pathology and Allergology.
• Republican Center for Hygiene, Epidemiology and Public Health.
• State Educational Institution "Belarusian Medical Academy of Postgraduate Education", Department of Gerontology and Geriatrics with a course of allergology and occupational pathology.
• Establishment of education "Belarusian State Medical University" (course of professional pathology).

Regional level of service of professional pathology:
• Regional center of occupational pathology based on health organizations of regional subordination.
• Chairs (courses) of occupational pathology of educational institutions "Vitebsk State Order of Peoples' Friendship Medical University", "Grodno State Medical University", "Gomel State Medical University".

• Occupational paths, which are part of the structural units of regional hospitals or other health organizations of the oblast subordination.
The urban (district, interdistrict) level of service of occupational pathology includes:
• Doctor - occupational pathologist.
• Cabinet of professional pathology.

The organizational-methodical, consultative-diagnostic and expert-rehabilitation body that coordinates the issues related to the health of workers and the prevention of occupational and allergic diseases in the Republic of Belarus is the Republican Center for Occupational Pathology and Allergology. The center consists of ambulatory consultative and diagnostic, inpatient occupational pathological and allergological departments, toxicological, allergic and immunological laboratories.

Tests 5, 6. **If an assumption arises about the occurrence of a working occupational disease, the treatment-and-prophylactic institution must issue the relevant documents and send the worker for an additional examination to the regional or republican center of occupational health within a period of not more than two months.**
An acute occupational disease **is established by a doctor of a polyclinic or hospital with the obligatory registration of an emergency notice in such cases.**
The diagnosis of *chronic occupational disease is* **established by** *medical consultation commissions (WCC)* **of regional and republican centers of occupational pathology.**
The diagnosis of a chronic occupational disease can also be established by AHC of any medical institution where there is a occupational physician who has undergone postgraduate training in professional pathology. In such cases, a representative of the territorial center of hygiene and epidemiology and a representative of the administration of the enterprise where the sick person works must necessarily participate in the meeting of the WCC.
In determining the occupational disease and assessing its consequences, the MEDNC is based on the list of occupational diseases approved in the Republic of Belarus, which is used in establishing a diagnosis, examination of working ability, consideration of issues related to compensation for damage to the health of the victim.
Only the Republican Center for Occupational Pathology and Allergology is entitled in some cases to recognize as occupational diseases those diseases that are not included in the list of occupational diseases approved in the Republic of Belarus.
Republican Center for Occupational Pathology and Allergology:
• provides organizational and methodological management of regional professional pathological centers,
• provides advice to the population,
• develops new methods for the diagnosis, treatment and prevention of occupational diseases,
• develops and organizes activities for the medical and social rehabilitation of sick and disabled people with occupational diseases,
• analyzes occupational morbidity in the Republic of Belarus,
• provides training and certification of doctors in occupational pathology and allergology.

Tests 7-15. **The level of risk of occupational pathology depends on the hygienic conditions at the workplace, the intensity and severity of the labor process, the presence of harmful factors that are potentially dangerous for the health of workers. This risk can be predicted taking into account the class of working conditions at each specific production.**
The following classes of production conditions are distinguished:
1st grade. **Occupational risk is absent. These are optimal working conditions that ensure the preservation of health and high working ability among workers in the workplace.**

Special labor protection measures are not required, since there is practically no risk of occupational pathology. Optimal working conditions are established only for the parameters of the microclimate and factors of the labor process.
2nd grade. **Negligible (tolerable) occupational risk. Complies with safe working conditions. They are characterized by such production factors, the levels of which do not exceed the limits of hygienic standards, and the possible changes in the functional state of the organism, arising under their influence, are restored during the regulated breaks or by the beginning of the next shift and do not have a negative actions in the near and distant period on the health of workers and their offspring.**
3rd grade. **The working conditions are characterized by such production factors, the levels of which go beyond the limits of hygienic standards and have an adverse effect on the worker's body and / or its maintenance. Harmful working conditions according to the degree of deviation of the parameters of production factors from hygienic standards and the severity of changes in the body of workers are divided into 4 degrees of harmfulness:**
• *1st degree* - low (moderate) risk. Working conditions are characterized by such production factors, the levels of which have deviations from hygienic standards and the impact of which causes functional changes in the body, recovering, as a rule, with a longer (than the beginning of the next shift) interruption of contact with harmful factors, increase the risk damage to health.
• *2nd degree* - medium (substantial) risk. Working conditions are characterized by such production factors, the levels of which have deviations from hygienic standards and cause persistent functional changes in the body, leading in most cases to an increase in production-related morbidity (which is manifested by an increase in morbidity with temporary disability and, first of all, those diseases which reflect the condition of the most vulnerable organs and systems for these harmful factors), the appearance of initial signs or light x (without loss of occupational disability) forms of occupational diseases that occur after prolonged exposure (often after 15 years or more).
• *3rd degree* - high (hard-to-bear) risk. Working conditions are characterized by such production factors, the levels of which have deviations from hygienic standards and lead to the development of, as a rule, occupational diseases of mild and moderate degrees of severity (with the loss of occupational disability) in the period of work activity, including increased incidence rates with temporary disability.
• *4th degree* - a very high (intolerable) risk. Working conditions are characterized by such production factors, the levels of which have deviations from hygienic standards and under which severe forms of occupational diseases can occur (with loss of general work capacity), there is a significant increase in the number of chronic diseases and high incidence rates disability.
4th grade - **ultra high risk to life. Working conditions are characterized by such production factors, the levels of which significantly exceed the limits of hygienic standards and the impact of which during the working shift (or part of it) can endanger the life of an employee, a high risk of developing acute occupational diseases, including severe forms. At the same time, work should be carried out in appropriate means of individual protection and with strict observance of the regimes regulated for this type of work and ensuring safety for the health of workers.**
Information about the degree of occupational risk allows for an objective, comprehensive hygienic assessment of working conditions, certification of workplaces in production and on this basis to manage the health of workers, provide motivated social protection and social insurance against occupational diseases.

Test 16. **Diseases encountered in professional pathological practice are divided into three main groups:**

Occupational diseases caused by the action of a certain harmful factor of the working environment. These diseases have a well-defined, specific clinical picture. These include pneumoconiosis, acute and chronic intoxication with lead, mercury, phospho-ororganic pesticides, etc.
Diseases resulting from exposure to harmful factors of the working environment, but in the clinical picture of which there are no specific manifestations that uniquely indicate the professional nature of this pathology. These are chronic bronchitis that occurs when working in a dusty environment, neurological disorders associated with the effects of vibration and noise, diseases of the musculoskeletal system during physical overstrain, etc.
Diseases that are not etiologically associated with adverse factors of production conditions, but that can occur in a more severe and severe form when exposed to such factors. Thus, in individuals with an initial allergic status, when exposed to the dust factor of the working environment, bronchial asthma often occurs.

Test 17. **Acute occupational disease arises from the impact of a harmful production factor in the course of employment for no more than one working day (shift). Acute occupational diseases do not include occupational accidents that occur regardless of the victim's professional activities.**

Test 18. **Chronic occupational disease is formed by prolonged, systematic exposure to the damaging factors of the production environment.**
Chronic occupational diseases also include the immediate and remote effects of acute occupational diseases (for example, persistent organic changes in the central nervous system after carbon monoxide poisoning).
The development of occupational diseases is possible after a long period of time after stopping work with harmful, hazardous substances and production factors (example: late silicosis, berylliosis, etc.).
Occupational diseases also include diseases in the development of which the occupational disease is a background or a risk factor (for example, lung cancer developed on the background of silicosis, asbestosis, dust bronchitis).

Test 19. **According to the list of occupational diseases approved in the Republic of Belarus, which is built on the etiological principle and contains the names of diseases in accordance with the International Classification of Diseases 10 revisions, as well as a description of harmful and dangerous factors of the working environment, the labor process, the impact of which can lead to the emergence of each specific occupational disease, the following seven groups of occupational diseases are distinguished:**
*Diseases caused by chemical factors***. Working with chemicals with toxic properties (nitric acid, ammonia, benzene and its derivatives, sulfur, perhydrol, mercury and its compounds, lead and its inorganic compounds, tetraethyl lead, etc., toxic chemicals used in agriculture, etc.).**
Diseases caused by exposure to industrial aerosols. **Working in conditions of high content in the ambient air of dust containing silica in free and bound states, particles of coal, graphite, soot, metals, organic and artificial mineral fibers, plastics, vegetable fibers, etc. may occur.**
Diseases caused by physical factors. **They may occur in people working with radioactive substances, sources of ionizing, electromagnetic radiation, lasers, exposed to intense industrial noise, vibration, etc.**
Diseases associated with physical overload and overstrain of individual organs and systems. **May occur when performing work associated with intense static-dynamic loads on the musculoskeletal system, local muscular overloads, being in forced posture, requiring high coordination of movements, stereotypical movements, overvoltage of the vocal apparatus, maintenance of patients of psychiatric institutions, etc.**

Diseases caused by exposure to biological factors. **May occur when working in contact with infected sick people and animals infected with biological material, products of animal and vegetable origin (processing of fur, skin, etc.), antibiotics, fungi producing antibiotics, protein-vitamin concentrates, fodder yeast, compound feed, etc. .**
Allergic diseases. **May occur during work related to substances and compounds of allergenic effects.**
Neoplasm. **They can occur during work related to the products of the distillation of coal, oil, shale, components of glass-fiber sizing oil, other chemical compounds that have carcinogenic properties, and sources of ionizing radiation.**

Test 20. **Highly efficient, partially or fully automated technologies with a high degree of occupational safety are increasingly being used in industry. New, highly effective means of collective and individual protection against harmful and dangerous factors encountered in industrial production have been developed. For this reason, we should expect a decrease in the harmful effects on the workers of the following factors of the production environment: noise, vibrations, physical overvoltage, chemical toxins, ionizing and non-ionizing radiation, etc. Indeed, in modern conditions, the risk of severe forms of occupational diseases has sharply decreased. Cases of professional pathology with erased clinical manifestations that occur in individuals for a very long time (at least 20 years) who have worked with the least intensive intensity of adverse factors of the working environment have become typical.**
The development of new production processes, primarily chemical and biotechnological, has led to the emergence of conditions for professional contact with chemical and biological substances that have extremely high toxicity and allergenicity.
At the same time, under the conditions of the environmental situation that has significantly changed in recent years, due to the increased release of toxic and allergenic substances into the atmosphere, it has become difficult to distinguish between the erased forms of certain occupational diseases and those that are not etiologically related to the workplace at the victim's workplace.
The widespread use of personal computers and the use of information networks to work led to a significant increase in the intensity of intellectual work, the creation of conditions for overstretching the central nervous system, a visual analyzer in combination with immobility, and being constantly in forced posture. Quite often, computer workers ignore sanitary and hygienic standards governing such work. As a result, combined occupational diseases of the nervous system, musculoskeletal system, cardiovascular system, and visual impairment occur.
In the process of periodic examinations, it is increasingly necessary to meet with signs of the nonspecific influence of various harmful factors of the production environment on the clinical course of chronic diseases that are not related to production.
In the professional pathological practice, a new phenomenon has emerged - "Social phobia",which is understood as a kind of psychological deviation, when the pathological fear of losing high wages, and subsequently the preferential pension for working under the conditions of the harmful factors of the working environment, makes an employee refuse recommendations on rational employment, which ultimately can lead to negative health effects, loss of professional ability to work.

Tests 21, 22. **An acute occupational disease is established by a polyclinic or hospital physician with mandatory registration of emergency notice in such cases. The diagnosis of acute occupational disease or poisoning can be established in all types of health facilities. The diagnosis of chronic occupational disease is established by VCC regional centers and the republican center of occupational pathology. If there is an assumption about the occurrence of a professional occupational disease in a health care institution, the institution**

must draw up the relevant documents and send it to the regional or republican center of occupational pathology for a period of not more than 2 months.
When referring a patient to medical institutions that have the right to establish the diagnosis of occupational disease, it is necessary to prepare the following documents:
• Direction of the medical institution.
• An extract from the medical card of the outpatient or inpatient where the following should be indicated:
- all transferred diseases;
- time of occurrence;
- results of special instrumental and laboratory research;
- list of therapeutic measures and their effectiveness.
• Information on the results of preliminary (upon receipt for work) and periodic medical examinations.
• Sanitary and hygienic characteristics of working conditions.
• Copy of employment record.
The methodology of clinical examination of patients and means of diagnosing occupational pathology have specific features, which is associated with the need to identify the etiological factor that caused the disease and, thus, to establish the causal relationship of the disease with unfavorable working conditions in the workplace of the victim.
To confirm the diagnosis of an occupational disease, a thorough analysis of the patient's history is made — drawing up of his professional route. Professional route allows you to establish not only the fact of contact with a harmful factor of the working environment, but also the duration (experience) of harmful effects. A sanitary and hygienic assessment of the working conditions of the sick person is mandatory. A study is performed on the functional state of various organs and systems in a patient. So, for example, to assess the nature of lung damage, determine the degree of dysfunction of respiratory function in individuals whose occupational disease is associated with work in dusty conditions, light radiography and a spirometry study are performed. In addition to general clinical examination, special tests are performed to identify specific signs of certain occupational diseases. For example, the detection of lead, mercury in the urine in the proof of professionally caused by intoxication with these metals.

Tests 24-29. At present, the Resolution of the Council of the Ministry of Foreign Affairs of the Republic of Belarus No. 30 of January 15, 2004 "On the investigation and registration of unfortunate cases at work and occupational diseases", regulating such work in the field.
Each identified or suspected case of *acute occupational disease* **within 12 hours should be sent a notice of acute occupational disease in the prescribed form to the employer, the insured at the place of work of the sick person, to the territorial center of hygiene and epidemiology. In the case of acute occupational diseases in case of simultaneous occupational disease of two or more employees, a notice shall be drawn up for each person who becomes ill. In cases of changes or clarifications of diagnosis, a second notification is made, indicating the modified or updated diagnosis, the date of its establishment, the initial diagnosis. This notice must be sent within 24 hours to the employer, the insurer, the territorial center of hygiene and epidemiology.**
In addition to sending a written notice, the employer, the insured, the territorial center of hygiene and epidemiology in each of the following cases should be immediately informed by phone, telegraph, fax, other means of emergency communication:
• acute occupational disease with a fatal outcome;
• acute occupational disease of two or more employees;
• diseases of anthrax, brucellosis, tetanus, rabies and other especially dangerous infections in connection with the professional activities of the victim.

The employer and the policyholder are also informed by the territorial prosecutor's office and the department of state labor inspectorate about acute occupational diseases with a fatal outcome, simultaneous occupational disease of two or more people. In such cases, the territorial center of hygiene and epidemiology provides an extraordinary report to the Ministry of Health.

In case of suspicion of a chronic occupational disease during a periodic medical examination, or when the employee personally refers in this regard, the health care organization prepares the necessary documents and establishes the final diagnosis in two months. If necessary, the sick person is sent for outpatient or inpatient examination to the relevant health care organization with the execution of the relevant documents.

In health care institutions that are competent to carry out the diagnosis of occupational diseases on the basis of clinical data on the state of health of the worker and the information presented in the accompanying documents, a final diagnosis is made of a chronic occupational disease, a medical report is drawn up and sent to the health care organization. sent the patient. And also, within five days, a notice is issued and sent to the territorial center of hygiene and epidemiology, the employer and the insured at the work place of the victim.

The investigation of acute occupational disease is carried out within three days, chronic - fourteen days from the date of receipt of the broadcast. It is carried out by the hygienist doctor of the territorial center of hygiene and epidemiology with the obligatory participation of:

• authorized officer of the employer;
• the insured;
• representatives of the healthcare organization;
• a representative of a trade union organization (or other representative body of workers).

To participate in the investigation are involved:

• specialists (upon request) required to investigate and assess damage in insurance claims;
• epidemiologist - when investigating cases of occupational diseases caused by especially dangerous and other infections;
• State Labor Inspector - when investigating occupational diseases of two or more people at the same time and cases of occupational diseases leading to death;
• specialists of higher centers of hygiene and epidemiology, research institutes - in the investigation of occupational diseases of two or more people and occupational diseases with a fatal outcome.

In the process of investigating an occupational disease, the following measures are taken:

• the workplace, the site, the workshop where the victim works are examined, their compliance with the requirements of sanitary and hygienic standards and the necessary laboratory and instrumental studies are determined;
• explanations are taken, the victim, witnesses, officials and other persons are interrogated;
• the provision of the victim with personal protective equipment, sanitary facilities and devices is established;
• documents on the results of sanitary and hygienic examinations, preliminary and periodic medical examinations, and implementation of planned labor protection measures are being studied;
• establishes the causes of occupational disease, persons who commit violations of legislative, regulatory and legal acts;
• technical, organizational, sanitary and hygienic, treatment-and-prophylactic, medical-rehabilitation and other measures are being developed to eliminate the causes and consequences of occupational disease.

According to the results of the investigation, the hygienist doctor draws up an act of a professional disease of a special form and makes copies of it in six copies, and in cases of

occupational disease of two or more people or a professional disease with a fatal outcome, in seven copies.
After approval of all copies of the act by the chief state medical doctor of the city (district), it is registered by the territorial center of hygiene and epidemiology in the register of occupational diseases and sent to:
• a sick person or a person representing his interests;
• the employer;
• the insured;
• the insurer;
• state labor inspector;
• health care organizations serving the employer, the insured;
• to the territorial prosecutor's office - in cases of occupational disease of two or more people and occupational diseases with a fatal outcome.
One copy of the act must be kept in the territorial center of hygiene and epidemiology.
Health care organizations that have departments of occupational pathology that conduct outpatient admission of patients of such a profile should keep a register of patients with occupational diseases.

Tests 30-32. **In accordance with the Resolution of the Ministry of Health of the Republic of Belarus No. 1/1 dated January 9, 2004, the MEDNC establishes individual rehabilitation programs for victims with occupational diseases who need medical, social and professional rehabilitation measures.**
An individual rehabilitation program serves as the basis for effective medical, vocational, and social rehabilitation aimed at restoring, compensating, and eliminating the limitations of occupational disability. MEDNEC fills out a special form, in which specific types, forms, volumes of the necessary rehabilitation measures, and the timing of their implementation are described:
Medical care: **taking into account the opinion of the WCT, the need for inpatient treatment of an occupational disease is indicated, with the definition of specific types of medical care.**
Supplementary food: **taking into account the opinion of the WCT, the need for a specific daily ration of additional food and the period for which it is prescribed is indicated.**
Drugs: **taking into account the opinion of the WCC, the need for specific drugs is indicated, indicating the duration and multiplicity of the courses of treatment and the period for which it is prescribed.**
Medical devices: **taking into account the opinion of the ICV, medical devices are listed, as well as the bedding that the patient needs.**
External care: **taking into account the opinion of the WCT, the need for special medical and / or domestic care is indicated.**
Sanatorium-resort treatment: **the need for sanatorium-resort treatment is indicated with prescription of the profile, frequency rate, season of the recommended treatment, as well as the need for an accompanying person.**
Prosthetics: **indicates the need for methods of reconstructive surgery and prosthetics; The type of prosthesis is indicated on the basis of the conclusion of the relevant specialists.**
Provision of equipment necessary for work and at home: **lists the technical means of rehabilitation (devices) necessary for work and at home.**
Provision of a special vehicle: **the conclusion of the MEDEC about the presence of medical indications to provide a vehicle with a corresponding management modification and the absence or presence of medical contraindications to its driving is recorded.**
Vocational training (retraining): **the necessity of acquiring another profession is established, indicating the form and type of training (retraining).**

Test 33. **Occupational diseases do not always entail a disability. At initial manifestations, mild clinical course of occupational disease, victims may be given recommendations to stop working in harmful and difficult working conditions according to the certificate of CWC. If there are grounds, patients are sent to the MEDNC, where they can be assigned the appropriate group of disability, the degree of loss of professional working capacity and the need for additional types of assistance are determined.**

Specialized professional pathological MREC is determined by:

• functional class of life restriction of the patient;

• reason, time of occurrence and disability group;

• degree of loss of professional working capacity (in percent);

• an individual program of medical, vocational and social rehabilitation, indicating the need for additional types of assistance (sanatorium-resort treatment, prosthetics, additional nutrition, the purchase of medicines, etc.).

When considering whether a disease is a professional, the specialized MEDNEC takes into account:

• the nature of the etiological factor acting on the patient and the work performed;

• features of the clinical form of the disease;

• specific sanitary and hygienic conditions of the working environment and labor process;

• Work experience in hazardous and hazardous working conditions.

In determining the occupational disease and assessing its consequences, the MEDNC is based on an approved list of occupational diseases.

In the case of a mixed etiology of occupational disease, expert questions about the cause of disability, the degree of disability are solved as a professional disease.

The violation of professional working ability **is the most frequent cause of social insufficiency - the inability of a person to perform a role that is usual for his social position. Occupational non-working capacity may arise primarily when other manifestations of a person's life activity are not violated. Or for the second time, on the basis of previously occurring impairments of vital activity caused by a general disease or an innate anatomical defect. Ability to work in a particular profession can be retained in persons with disabilities in whole or in part, and in some cases can be restored by measures of vocational rehabilitation. In such cases, people with disabilities can work in normal or specially created working conditions.**

The definition of the degree of loss of professional working capacity is carried out by MEDEC on the basis of the Resolution of the Council of Ministers of the Republic of Belarus No. 1299 of 10.10.2003 and the Resolution of the Ministry of Health of the Republic of Belarus No. 1/1 of January 09, 2004. In accordance with these documents, the degree of loss of professional disability is determined taking into account professional abilities, psycho-physiological capabilities and professionally significant qualities that allow the patient to continue to perform professional activities prior to the same content and in the same volume, or taking into account the reduction of qualification, the reduction of the volume of work performed and the severity of labor in ordinary or specially created conditions.

The degree of loss of professional working ability is expressed in pro-cent and can be set in the range from 10 to 100%.

Examination of professional disability is carried out on the basis of the assessment:

• Clinical and functional criteria.

• Ability to professional activity.

• Degree of loss of occupational ability.

Clinical and functional criteria include:

• the nature and severity of the occupational disease;

• features of the pathological process caused by a professional disease;

• nature of violations of the body;

• the degree of impairment of body functions (pronounced, severe, moderate, mild);
• psycho-physiological features of the patient;
• clinical and rehabilitation prognosis;
• clinical and labor prognosis.
Ability to professional activity **is assessed taking into account the professional factor - the ability after the occurrence of a professional disease to perform work in full according to their previous (before illness) profession or other qualifications equivalent to it.**
Professional activity in full involves a full day, full time, the implementation of standards for at least 100%.
Criteria for assessing the ability to carry out professional activities are directly related to the difference in tariff and qualification characteristics in the framework of the relevant profession. The multiplicity of decline in qualification is determined taking into account the establishment of qualification ranks, classes, categories for the profession.
When determining the degree of loss of professional working capacity of qualified workers, classes of working conditions are taken into account according to indicators of hazard and danger of factors of the working environment, severity and intensity of the labor process.
Ability to professional activity **is assessed taking into account the professional factor - the ability after the occurrence of a professional disease to perform work in full according to their previous (before illness) profession or other qualifications equivalent to it.**
Professional activity in full involves a full day, full time, the implementation of standards for at least 100%.
Criteria for assessing the ability to carry out professional activities are directly related to the difference in tariff and qualification characteristics in the framework of the relevant profession. The multiplicity of decline in qualification is determined taking into account the establishment of qualification ranks, classes, categories for the profession.
When determining *the degree of loss of professional working* **capacity of qualified workers, classes of working conditions are taken into account according to indicators of hazard and danger of factors of the working environment, severity and intensity of the labor process.**
The degree of loss of professional working ability of a worker with unqualified physical labor is established depending on his psychophysiological state, physical ability to perform simple physical work and is also associated with the assessment of classes of working conditions according to the severity of labor.
The degree of loss of professional working ability is estimated in percents based on the following regulated criteria:
1. **If, as a result of an occupational disease, the victim has a complete loss of professional disability due to a pronounced impairment of body functions, including in specially created working conditions, the degree of professional disability is set to 91 to 100 percent.**
2. **In the event that a victim as a result of a pronounced violation of the functions of the organism can perform work only in specially created production conditions, the degree of loss of professional working ability is established from 61 to 90 percent. Including:**
• if a victim who has previously performed qualified work in normal working conditions can only be performed by unskilled types of work in specially created working conditions, the degree of loss of professional working ability is set to 90 percent;
• if the victim can perform lower-skilled work in the specially created production conditions, taking into account professional knowledge and skills, the degree of loss of professional working ability is set to 80 percent;
• if a victim can perform work in a profession prior to an occupational disease in specially created conditions, the degree of loss of professional working ability is determined from 61 to 70 percent.
3. **If the victim can continue his professional activity under normal production conditions with a marked decline in qualifications, or with a decrease in the amount of work performed or if he has lost the ability to continue his professional activity as a result of**

moderate disruption of body functions, but may under normal production conditions professional activities of lower qualifications, establishes the degree of loss of professional ability to work from 25 to 60 percent. Including:
• if the victim has lost his profession and can perform light unqualified types of work, or can perform work in the profession, but with a decline in qualification for four rating levels, or can perform unskilled physical labor with a reduction in the number of jobs into four categories of gravity, establishes the degree of loss of professional disability up to 60 percent;
• if the victim can perform work in a profession with a decrease in qualification by three rating classes, or can work in a profession with a decrease in the volume of production activity (by 0.5 rates), or can perform unskilled physical work with a decrease in the category of work into three categories severity, establishes the degree of loss of professional working ability to 50 percent;
• if the victim can perform work in a profession with a decrease in the volume of production activity, or work not in a profession, but using professional skills, or work in a profession with a reduction in qualification by two rating classes, or unskilled physical work with a decrease in the category of work by two the category of gravity, establishes the degree of loss of occupational ability from 25 to 40 percent.
4. **If the victim can continue his professional activity with a moderate or insignificant loss of qualifications, or with a decrease in the amount of work performed, or when working conditions change, entailing a decline in earnings, or if the performance of his professional activity requires more stress than before The degree of loss of professional working ability is from 10 to 24 percent. Including:**
• if the victim can perform work in a profession with a decrease in qualifications for one rating grade, or unskilled physical work with a decrease in discharge of work per category of severity, or work in the main profession with a slight decrease in the volume of professional activity (decrease in the rate of output by 1/3 of the former load), establishes the degree of loss of professional ability to work - 24 percent;
• if the victim can perform work with a decrease in the volume of professional activity by 1/5 of the previous load, the degree of loss of professional disability is set to 20 percent;
• if the victim can perform work with a decrease in the volume of professional activity by 1/10 of the previous load, the degree of loss of professional working ability is set to 10 percent.
The degree of loss of professional working ability is not determined, if a professional disease diagnosed during the period of employment did not result in the loss of the victim of professional work ability during the period of employment, it allowed him to work in the relevant profession without loss of qualifications, class of work up to retirement age and at the time of the survey does not limit the ability to all types of employment.

Test 34. **Occupational diseases can cause temporary, long-term or permanent disability. Temporary disability is determined by IHC for not more than 4 months (120 days) without a break or 5 months (150 days) with a break. As a rule, this happens with severe acute intoxication, with exacerbation of chronic occupational diseases.**
Long-term or permanent loss of working capacity or its significant limitation can be established only by MEDN with the definition of the group of disability. When determining the disability group, MEDNC uses the criteria "Instructions on the procedure and criteria for determining the group and cause of disability, the list of medical indications giving the right to receive a social pension for disabled children under the age of 18, and the degree of loss of their health », Approved by the Decree of the Ministry of Health of the Republic of Belarus No. 97 of October 25, 2007

Tests 35-39. **In accordance with the Instruction, MEDNEC conducts a quantitative assessment of the degree of loss of function and disability. There are five functional classes:**
• FK-0 - no impairment of function and disability.
• FC-1 - a slight disability due to the loss of up to 25% of functions.
• FC-2 - moderate limitation of vital activity due to the loss of 26 to 50% of functions.
• FK-3 - significant limitation of vital activity due to the loss of 51 to 75% of functions.
• FK-4 - a pronounced limitation of vital activity up to the complete loss of functions - 76-100%.

Test 40-42. **Disability is determined in case of occurrence of violations of professional working capacity based on the following criteria:**
Group I disability **is established with complete disability, when the patient, due to the presence of pronounced functional disorders, needs constant assistance, care or supervision and corresponds to FC-4.**
Group II disability **is established for patients with FC-3, who:**
• labor is not available due to severe functional limitations caused by the disease;
• labor is contraindicated due to the worsening of the patient's condition as a result of any professional work activity;
• labor is not contraindicated, but is available in specially created conditions.
Group III disability **is established in patients with FC-2 in accordance with the following criteria:**
• in case of a forced transfer due to the state of health to a lower qualification (decrease in qualification by 4 grades or more, reduction in category, management level for managers);
• with a decrease in health standards, production, reduction for the same reasons, the duration of the working day.
In combination with a general disease, in which disability group II is established, with an occupational disease, in which disability group III is established, the patient is recognized as a disabled person in group II due to an occupational disease.

Test 43. **A re-examination of persons with disabilities in group I is carried out every 2 years, and groups II and III - after 1 year.**
The third group of disability is usually established for a period of no more than 1-2 years, which, as a rule, is necessary for vocational rehabilitation - re-training and rational employment of the patient.
Re-examination of the previously mentioned deadlines is carried out in case of deterioration of the health status and degree of disability of the disabled person. As well as at the opening in the process of preliminary investigation of the facts of illegal actions (false documents, etc.).
Disability without a deadline for re-examination is established:
• women over 55 and men over 60;
• with persistent, irreversible morphological changes and dysfunctions of organs and body systems of a disabled person;
• impossibility of improving the course of the disease and social adaptation due to the ineffectiveness of the rehabilitation activities carried out after 3 years of MEDN monitoring;
• with persistent anatomical defects listed in a special list of Instructions for determining disability groups.

Test 44. **Compensation for damages in connection with the disability caused by an occupational disease is determined by the MEDEC since the moment of diagnosis, but no more than three years from the date of treatment of the victim for compensation for harm.**

Patients with occupational pathology enjoy additional benefits. With temporary disability, sick leave is paid by them in the amount of 100% of earnings, regardless of the length of service.
For the period of examination in the centers of occupational pathology, a certificate is issued to patients, on the basis of which the average salary is maintained.
The size of the disability pension is also assigned regardless of the length of service in hazardous conditions that caused the illness resulting in disability. The amount of pensions for professionally determined invalidity is greater than the size of pensions for general disability, since co-payments are made to this pension depending on the percentage of loss of professional working capacity established by the MEDEC.
Individuals with occupational diseases are provided with free trips to a sanatorium. Such patients are entitled to additional material compensation on the part of the employer if the professional illness has arisen solely through the fault of the employer.

Test 45. Medical examinations that are preliminary at the time of entry into work **allow for the selection of persons who are not contraindicated in working with a particular production factor due to their physical development and state of health. Preliminary examinations are carried out only on the direction of the employer, which indicates the name of the profession and occupational hazards in this particular production, general and additional contraindications to employment. In the absence of these data, a medical examination is not carried out and a validity report is not issued.**

Test 46. **The results of the preliminary medical examination are documented in the form of a socio-clinical conclusion. On the basis of the materials of this conclusion, a certificate is issued to the person being examined with a reference to "fit" or "unfit" to perform this work without detailing the results of the examination.**

*Tests 47, 48. Periodic medical examinations provide***:**
• dynamic monitoring of the health of workers under the influence of occupational hazards;
• identification and prevention of the initial signs of occupational diseases;
• identification of common diseases in which further work under conditions of exposure to occupational hazards may worsen their clinical course;
• assessment of working conditions with the subsequent development of sanitary and hygienic measures to improve them;
• preventing the spread of infectious and parasitic diseases.
The following categories of persons are subject to periodic medical examinations:
• working under the influence of harmful and (or) dangerous factors of the production environment, indicators of the severity and intensity of the labor process;
• employees of catering and food industry; dairy farms, as well as at work, where there is contact with food products;
• students of secondary and higher educational institutions;
• employees of educational, preschool, health institutions;
• workers of housing and communal services;
• medical workers;
• workers of water supply facilities associated with the preparation of water and maintenance of water supply networks;
• workers of all types of transport associated with the direct service of passengers.
The data of the periodic examination are recorded in the medical card of the ambulatory patient. In addition, each doctor participating in the examination, gives his individual conclusion about the professional fitness of the employee. The data of the professional

route of the employee: enterprise, workshop, site, profession, harmful and unfavorable working conditions are entered on a separate sheet of the ambulatory card.
After the inspection, a final report is drawn up in 3 copies, one of which remains in the medical institution that conducted the medical examination, and the other two are sent to the employer at the territorial center of hygiene and epidemiology.
The act records the results of the medical examination in accordance with the form of the Annex to the *«Instruction on the procedure for conducting mandatory medical examinations of workers»* **, approved by Resolution of the Ministry of Health of the Republic of Belarus No. 47 of April 28, 2010. It also indicates:**
• implementation of sanitary and hygienic measures provided for by the previous act;
• lists of workers who have not undergone a physical examination, indicating the reason;
• lists of workers with suspected occupational disease;
• lists of workers requiring an extraordinary medical examiner;
• lists of persons with common diseases that have been identified that interfere with and do not prevent the continuation of work;
• a list of employees who are assigned to conduct therapeutic and recreational activities.

Test 49. **The organization and conduct of preliminary and periodic medical examinations is carried out on a contractual basis with the subjects of the treatment and prophylactic institutions. The commission, as a rule, consists of the chairman and members of the commission: at least three doctors-specialists of the healthcare organization, as well as a psychiatrist-narcologist and a hygienist (in consultation with their leaders).**
When a general practitioner is included in the commission if there are no other specialist doctors in the commission, it is allowed to conduct a medical examination of the employee and make a decision about the absence (presence) of diseases that impede work in this profession.
The commission is headed by the chairman of the commission, a specialist who has the qualification of a "doctor-professional pathologist". Commission members should be trained on issues of occupational pathology.
To conduct periodic medical examinations of employees under employment contracts, an employer who provides employment under an employment contract annually draws up a list of occupations (positions) of employees subject to periodic medical examinations in the form in accordance with the Appendix to the Instruction.
Periodic medical examinations of workers not included in the list of occupations are carried out on the basis of the direction of the employer to the health care organization.
The list of occupations is compiled taking into account the results of a comprehensive hygienic assessment of working conditions, results of certification of workplaces for working conditions, harmful and (or) dangerous factors of the working environment, indicators of the severity and intensity of the labor process specified in Appendix 1 to the Instruction, and works specified in Annexes 2, 3 of the Instructions.
The list of occupations is sent to the health care organization until January 1 st, during which it is necessary to conduct a scheduled periodic medical examination (hereinafter referred to as the current year).
The organization of health care on the basis of the list of professions shall be drawn up and sent to the employer no later than February 1 of the current year schedule for conducting periodic medical examinations.
The employer, on the basis of the list of professions and the schedule for conducting periodic medical examinations, draws up a list of employees subject to periodic medical examination, in accordance with the annex to the Instruction.
The list of workers 15 days before the start of the periodic medical examination is sent to the health care organization. Periodic medical examinations of workers are completed before December 1 of the current year.

The timing of periodic medical examinations depends on the class of working conditions, type of production and the profession of the employee. But in any case, they should be held at least 1 time in 3 years. The specific periodicity of medical examinations, the participation of certain specialists, the necessary laboratory and functional studies are determined in accordance with the requirements set forth in the annexes to the "Instructions on the procedure for conducting mandatory medical examinations of workers" (approved by Healthcare of the Republic of Belarus of 28.04.2010, the number 47). The composition of specialties of doctors participating in medical examinations, the scope of diagnostic studies, medical contraindications to work permit are regulated in 1, 2, 3 annexes to the Instructions.
Doctors participating in the preliminary examinations get acquainted in detail with the professional route, history, discharge from the outpatient card about the diseases suffered, military ticket. They conduct a thorough general examination,, study the data of laboratory and instrumental studies. Special attention is paid to the state of those organs and systems that are the most vulnerable (critical) under the influence of occupational hazards. When hiring people who are registered with the TB dispensary, a TB doctor is required.
Doctors of various specialties participate in periodic medical examinations in accordance with the established list. As a rule, these are doctors who have the specialty of a therapist, a neuropathologist, an ophthalmologist, or an otorhinolaryngologist. If necessary, other specialists are involved - allergists, dermatologists, phthisiatricians, infectious disease specialists, toxicologists, orthopedic surgeons, etc.
The complex of compulsory studies includes: complete blood count, mochi, biochemical blood tests, chest X-ray, ECG, ultrasound. Additionally, if there is a need, studies are conducted on the presence of heavy metal ions in the urine (mercury, lead), the study of the content of methemoglobin, pseudocholinesterase in the blood, coproporphyrin and delta-aminolevulinic acid in the urine, the study of respiratory function etc. Other methods are also used to identify specific signs of certain types of occupational diseases.
According to the totality of data obtained during the medical examination and taking into account the results of laboratory and instrumental studies, the doctors who took part in the examination, draw up a medical report and determine, if necessary, a set of treatment-and-prophylactic or medical rehabilitation measures for each examined person.

Test 50. **The general contraindications to admission to work related to harmful and hazardous substances and unfavorable factors are any chronic diseases in the presence of even moderately pronounced insufficiency of the function of internal organs, and in women - the state of pregnancy and lactation.**
In addition to general contraindications, additional ones have been developed - for each harmful production factor, taking into account the specific features of its effect on the human body. Thus, at the enterprises for the production and use of chemicals of hazard class I, female labor is prohibited. Women working in the conditions of harmful production, from the day of pregnancy, are transferred to work outside the influence of adverse factors. Persons under the age of 18 are prohibited to work in hazardous conditions, underground, and also associated with great physical exertion.
The following criteria are used in selecting individuals who are contraindicated in employment for allergic hazardous work:
*Burdened hereditary immunoallergological history***. It occurs when there are two generations of allergic, autoimmune, primary immunodeficient, chronic infectious-inflammatory diseases, and malignant neoplasms in the family and in close relatives.**
Burdened own allergic history. **Allergic reactions to prophylactic vaccinations, medication, use of cosmetics, contact with chemicals used in everyday life, insect bites are taken into account. Information about previous professional contacts with substances with toxic-allergic properties is important.**

Retrospective medical contraindications. **Anamnestic information indicating the presence of chronic immune deficiency, manifested by recurrent, chronic, low-intensity infectious processes of various localization, is taken into account.**

Objective clinical contraindications. **There are three groups of such contraindications:**

.*1 General contraindications***. This includes any illnesses from the listed contraindications to work in hazardous and hazardous working conditions on the official list.**

.*2 Absolute contraindications***. This includes all primary and secondary immunodeficiency diseases. The list of these diseases is set out in a special list of contraindications to the permit for allergic hazardous work.**

.*3 Relative Contraindications***. The presence of acute and sub acute infectious and inflammatory diseases (not included in the official list of contraindications) that can worsen or become more severe during the production of allergens.**

*Impaired immune status***. They are established if, when performing non-specific and specific sensitive laboratory samples, provocation tests with allergens from the working**

EXPLANATION TEXTS
to the test tests section II
“PROFESSIONAL DISEASES OF RESPIRATORY ORGANS CAUSED BY EXPOSURE TO INDUSTRIAL AEROSOLS”

Test 1. **Occupational lung diseases caused by dust include pneumoconiosis, byssinosis, chronic occupational dust bronchitis. According to the approved list of occupational diseases, occupational bronchial asthma resulting from inhalation of allergenic dust belongs to the group of allergic diseases.**

Tests 2-5. **Human inhaled dust can penetrate into the airways at different depths, depending on the size of the dust particles. Large particles that are larger than 20 microns, are deposited on the mucous membrane of the upper respiratory tract, large bronchi. Dust size of about 10 microns can deposit on the surface of the middle and small bronchi. If the size of the dust particles does not exceed 5-7 microns, they are able to reach the alveoli. However, they cannot settle in normal alveoli, since the electrostatic charge on the wall of the alveoli creates a repulsive force that exceeds the gravity of these particles.**

Tests 6-9. **Pneumoconiosis is a common name for chronic occupational diseases with damage to the respiratory organs caused by prolonged exposure to industrial dust, which leads to fibrosis of the lung and pulmonary insufficiency.**
There are 6 main groups of pneumoconiosis, depending on the composition of the dust that caused the occupational disease:
• Silicosis - from breathing in dust containing free silica (silica dust).
• Silikatoza - from inhalation of silica hydroxide dust (silicates):
- **asbestosis - from inhaling asbestos dust;**
- **apatitis - from breathing in apatite dust;**
- **talcosis - from breathing in talcum dust;**

• Carboconiosis - from breathing in dust containing free carbon:
- **anthracosis - from inhalation of coal dust.**
- **graphitosis - from inhalation of graphite dust.**

• Metalloniozy - from inhalation of dust containing metals and their water-insoluble salts:
- **siderosis - from inhalation of iron dust;**
- **aluminum - from breathing in aluminum dust.**
- **baritosis - from inhaling the dust of barium compounds.**
- **manganoconiosis - from inhalation of manganese dust.**

• Mixed - from inhalation of multicomponent inorganic dust:
- **siderosilicosis - from inhalation of dust containing free silica and iron.**
- **silikoantrakoz - from inhalation of dust containing free silicon dioxide and coal.**
- **pneumoconiosis of electric welders.**

• Evolving from inhaling organic dust:
- **amylose - from inhalation of flour dust.**
- **tobacco - from inhalation of tobacco dust.**
- **Farmer's lung - from breathing in dust of hay, straw, containing fungus.**

Test 10. **The fibrogenic activity of various types of dust depends on the percentage of free silica in the dust.**

Tests 11, 12. Currently, all pneumoconiosis is divided into three groups according to the ability of various types of dust to activate the processes of fibrosis in the lungs or to cause toxic-allergic reactions.

Pneumoconiosis resulting from the inhalation of high or moderate but fibrogenic dust. **These include primarily silicosis, as well as pneumoconiosis from inhalation of mixed dust containing more than 10% of free silica (silicoanthracosis, siderosilicosis), with the exception of pneumoconiosis caused by radiopaque dust (siderosis, manganoconiosis, pneumoconiosis of welders). Pneumoconiosis from high-fibrogenic dust is characterized by the formation of granulomatous inflammation with a transition to pulmonary fibrosis.**

Pneumoconiosis, resulting from the entry into the respiratory tract of low-fibrogenic dust, which has in its composition less than 10% free silicon dioxide or does not contain it at all. **This group includes all sil-cathosis (asbestosis, talcosis, apatitis), carboconiosis (anthracosis, graphitosis). These are the most common lung dust lesions. Differ in slowly progressive and usually moderately severe pulmonary fibrosis, rarely leading to fatal decompensation of lung function. Complicated diseases can contribute to the worsening of the patient's condition: obstructive dust bronchitis, emphysema with changes in the lungs of a restrictive type (the distension of the lungs is disturbed during inhalation).**

Pneumoconiosis from inhalation of dust particles that do not have fibrogenic properties, but have a toxic-allergic effect. **Such pneumoconioses can be caused by dust of non-ferrous metals-allergens, plastics, organic dust (farmer's lung). Autoimmune mechanisms of formation of hypersensitive alveolitis can be activated by inhalation for a short period of time of a large volume of aerosols, which have a current-allergic effect. This can happen in case of violation of safety procedures - performing welding and casting works with metals such as copper, tin, zinc, tungsten, etc. in enclosed areas in the absence of adequate ventilation with the formation of a clinical picture of so-called "foundry" fever. Toxic-allergic diseases of the lungs can occur in conditions of exposure to dust particles, even if there is no excess of their maximum allowable concentration in the air we breathe. Occupational lesions of the respiratory system of this type in the initial stages of the disease are accompanied by bronchiolitis and alveolitis, with further transformation into diffuse pulmonary fibrosis. A typical form of toxic and allergic lesion of the lungs is berylliosis.**

Tests 13, 14. **With silicosis, dust particles ranging in size from 7-10 microns and more are deposited on the bronchial mucosa. Dumps with sizes less than 5-7 microns can penetrate into the alveoli, but in the absence of obstacles to the movement of air through the small bronchi, they leave from there. Surface-active polarized substances - surfactants, create an electric charge that prevents the deposition of small particles that have penetrated the alveoli, including bacteria, fungi. When working in dusty conditions, regardless of the type of dust, endobronchitis is sure to occur. With a long course of endobronchitis, the ciliated bronchial epithelium atrophies. The drainage function of the bronchial mucosa is impaired. Periodically occurring obstructions of the small bronchi cause microatelectasis, ensuring the contact of the wall of the collapsed alveoli with small particles of silicon dioxide.**

Dust microparticles from the surface of collapsed alveoli are captured by alveolar macrophages and with their help are transported into the lymphatic capillaries of the interstitial tissue of the lungs.

Crystals of quartz dioxide, especially with fresh fractures, have the ability to chemically combine through silane groups with proteins. Protein-silicon structures are perceived by the immune system as foreign antigens. However, the enzyme systems of macrophages that have absorbed such antigens are not able to destroy the crystals of silicon dioxide. Macrophages die. The microcrystals of silicon dioxide released from the dead macrophages are again phagocytosed by other macrophages, which also die, being unable to destroy them. In order to isolate particles of silicon dust, a protective immune barrier in the form of a granuloma is formed around them. Inflammatory mediators released at the location of

the granuloma, activate the processes of fibrogenesis, with the result that the granuloma is later replaced by fibrous tissue. Granulomatous-fibrous foci give a characteristic X-ray picture of lung nodular lesions.
The type of granulomatous reaction depends on the type of dust. When breathing highly fibrinogenic dust containing more than 10% free silica, granulomas consist mainly of macrophages. When exposed to dust with toxicoallergic properties, granulomas are formed from epithelial cells.

Test 15. **With silicosis, silicotic nodules form in the alveoli and alveolar passages. They can also occur in the perivascular and peribronchial spaces along the lymphatic vessels.**

Test 16. **There are two variants of the morphological structure of the silicotic nodules. One option, which is considered the most typical, has the correct round or oval shape. Such nodules consist of concentrically arranged layers of partially hyalinized connective tissue. Another, less typical variant of silicotic nodules has an irregular shape. The bundles of connective tissue that make them are randomly arranged.**
In accordance with the size, the silicotic nodules are subdivided into submillion, miliary and large ones. Nodules can merge into fibrous fields. Necrotic changes often occur in large nodes. Lime deposits are deposited in place of the necroses, and silicotic caustics are formed.

Tests 17-21. **When working in conditions of extremely high content in the inhaled air, silicon dioxide dust during the year can form "acute" silicosis. It is characterized by persistently progressive course, rapid, within 1-3 years, the formation of massive pulmonary fibrosis. Currently, due to the improvement of safety at work and for this reason, the lack of conditions for its occurrence, this variant of silicosis is not found.**
Silicosis can occur in the following forms:
• Rapidly progressive silicosis. According to the clinical course is close to "acute" silicosis. It develops in 3-5 years after the start of contact with silica dust. Transitions from the initial to the subsequent stages of the disease occur in 2-3 years.

• Slowly progressive silicosis. It develops with long-term inhalation of dust with a relatively low content of silicon dioxide for 10-12 years. In the initial stages, it usually proceeds clinically latently. Transitions between stages of the disease last 5-10 years.
• Late silicosis. Occurs several years later after the termination of the contact with silicon dust. It is characterized by progressive fibrosing lesions of the lungs, respiratory failure.

Tests 22-24. **In the clinical development of silicosis, there are three stages, each of which corresponds to certain pathological changes in the lungs.**
Stage I of silicosis **corresponds to diffuse sclerotic changes in the lungs. At this stage, the clinical manifestations are very scarce. Patients may complain of a rare dry cough, chest pain, shortness of breath during physical exertion.**
An objective examination can not detect anything. Only in some cases, with percussion of the chest, the expansion of the roots of the lungs is detected, and harsh breathing is heard.
An X-ray examination can reveal a bilateral diffuse amplification characteristic of this stage, a deformation of the bronchopulmonary pattern. Symmetric expansion, compaction, deformation of the roots of the lungs is determined. The shadow of the heart is usually not extended.
Deviations of laboratory, biochemical indices of blood, urine, pathological changes on the ECG, during echocardiography, are usually not possible to register.
Stage II silicosis **corresponds to the nodular form of the disease. At this stage, inspiratory dyspnea becomes noticeable in patients. Concerned about soo-hoi cough, chest pain.**

Objective data are usually the same as in stage I of silicosis. Sometimes the pleural friction noise can be heard. A boxed shade of percussion sound may appear above the lower side sections of the lungs.
X-ray examination revealed more pronounced changes in the form of compaction, expansion, deformation of the roots of the lungs, increased bronchopulmonary pattern, signs of basal emphysema. Against this background, there are nodular changes, symmetrically scattered throughout all fields, somewhat more in the middle and lower parts of the lungs. All nodules are usually the same size and density. Their diameter can be from 1-2 mm to 18-10 mm. Usually detected thickening and deformity of the pleura.
In the study of respiratory function, signs of pulmonary insufficiency of grade I-II are detected, mainly by restriction type.
Stage III silicosis **corresponds to massive, large-nodal fibrosis of the lungs. Despite the gross changes in the lungs, the general well-being of the patients at this stage of the disease can remain satisfactory. Disturbing mainly shortness of breath with difficulty inhaling, at least - inhalation and exhalation, dry or unproductive cough with chest pains. The increase in the severity of the clinical manifestations of the disease is usually associated with decompensation of the chronic pulmonary heart. In such cases, peripheral edema appears, the liver increases, ascites develops.**
With an objective study of all the fields of the lungs, percussion sound with a box tint, pleural friction noise, non-sound crepitations, indicating the formation of common emphysema, dry pleurisy, and pneumosclerosis. Expand the boundaries of the heart. Heart tones are muffled. II tone above the pulmonary artery is accentuated. The liver is enlarged. Pasteal thighs. With decompensation of the pulmonary heart, peripheral edema and ascites are detected.
On ECG and EchoCG, signs of hypertrophy and dilatation of the right ventricle and atrial myocardium are detected.
On radiographs in the lungs, asymmetrically located large foci (nodes) of various sizes are visible, forms against the background of gross structural changes, the same as in stage II of silicosis. At the same time, there is a thickening and deformation of the pleura, interpleural adhesions, signs of bullous emphysema, in some cases - silicic caverns, having a slit-like shape, not containing a liquid level, having calcareous inclusions in their contour are detected.
In the study of respiratory function, signs of pulmonary insufficiency of grade II – III are determined by restrictive or mixed type.

Tests 25-28. **Tuberculosis usually develops against the background of severely flowing forms of silicosis, in the presence of pronounced immunity disorders.**
With a complication of tuberculosis, the clinical picture of silicosis substantially changes. There is unmotivated general weakness, fatigue, sweating, cough increases. Appears not characteristic of uncomplicated silicosis subfebrile. In general, as well as biochemical blood tests, abnormalities can be recorded, indicating the existence of an active inflammatory process in the lungs. The results of skin tuberculin tests are becoming positive. The rapid progress of pathological changes in the lungs appears. The X-ray picture of changes in the structure of the lungs acquires polymorphism and asymmetry, which is unusual for silicosis.
Latent flowing silicotuberculosis lymphadenitis is manifested by shell-like calcification of mediastinal lymph nodes. Infiltrative pneumonic silicotuberculosis is accompanied by the appearance in the lungs of rounded cloud-like segmental or occupying a whole proportion of infiltrates. Hematogenically disseminated tuberculosis on the background of lycotic changes in the lungs has a peculiarity - polymorphic dissemination foci are located mainly in the upper parts of the lungs. Fibrous-cavernous silicotuberculosis is characterized by the appearance of light-rounded caverns or scalloped focal formations. Perhaps the formation

of a silicotuberculosis. Such a variant of silicotuberculosis is usually accompanied by clinical and laboratory symptoms of an active inflammatory process.
With silicotuberculosis, hemoptysis occurs extremely rarely, and in the wet, tuberculosis mycobacteria usually cannot be detected.
Test 29. Silicarthritis or Kaplan syndrome is a combination of the most often interstitial or nodular form of silicosis with rheumatoid arthritis. Quite often, when silicoarthritis in the peripheral parts of the lungs, round infiltrative formations of 0.5 to 2 cm in diameter are detected. Lesions of the joints do not differ from those arising from classical rheumatoid arthritis. Arthritis begins with a symmetrical bilateral lesion of the small joints of the hands. Less commonly, the pathological process begins with a symmetrical lesion of other joints. Patients are concerned about morning or constant stiffness, arthralgia, swelling, restriction of mobility of the affected joints. Radiographs reveal uzura articular surfaces - typical for rheumatoid arthritis signs of erosive ankylosing lesions of the joints.

Tests 30-31. **Silikatoza are pneumoconiosis, resulting from exposure to the respiratory tract of weakly fibrogenic dust of silicates, which has less than 10% of free silica in its composition. The main component of such dusts is silicon hydroxide and its compounds - silicates. Natural silicates are the minerals asbestos, talc, nepheline, kaolin, mica, olivine. Artificial silicates are glass, cement. Silicates are widely used in industry. Therefore, silicates are one of the most common occupational dust diseases of the lungs. They are characterized by slowly progressive and, therefore, usually moderately severe fibrosis, rarely resulting in fatal de-compensation of lung function. The occurrence of obstructive dust bronchitis and pulmonary emphysema contributes to the condition of patients with professional silicatosis.**

Test 32. **Silicate - asbestosis is a disease caused by the inhalation of asbestos dust - a natural fibrous mineral. In the industry, chrysotile asbestos is used as a heat-resistant insulating material, consisting of a complex compound of silicic acid with salts of magnesium, aluminum, and iron. This mineral usually contains small admixtures of free silica.**
In addition to the moderate fibrogenic effect, asbestos dust has a pronounced irritant effect on the respiratory tract. Changes in the lungs are characteristic of asbestosis in the form of diffuse fibrosis with the involvement of peribronchial, perivascular interstitial tissue, interlobular and alveolar septa. Asbestos bodies form in the lungs. There are numerous pleural adhesions. In compacted bifurcation and primitive lymph nodes accumulate a large number of asbestos dust particles delivered by macrophages through the lymphatic capillaries. In asbestosis, there are no nodules in the lungs that are typical of highly fibrogenic silicosis. Unlike other pneumoconiosis, asbestosis very often forms bronchiectasis. Asbestosis can cause the formation of squamous cell carcinoma from the epithelium of the mucous membrane of the small bronchi.

Tests 33-35. **The clinical picture of asbestosis is formed from the symptoms of chronic dust bronchitis and pulmonary fibrosis. Asbestos fibers and bodies are detected in sputum. Asbestos bodies are also found in the histological preparations of the lung tissue — linear microstructures with thickened ends in the form of weights, drum sticks — modified particles of asbestos fibers. Asbestos warts may occur on the skin - an inflammatory-proliferative reaction to the introduction of asbestos fibers into the epidermis.**
For *stage I of asbestosis*, **complaints of dyspnea during physical exertion, dry cough, and chest pain are characteristic. Disturbed by general weakness, increased fatigue. Patients lose weight. The skin has a grayish-earthy tint with a cyanotic hue. Physical examination determines the percussion signs of emphysema. The pleural friction noise, scattered dry rales over the lower side of the lungs are heard. Radiographically determined moderate compaction, root deformity, reticular enhancement of the pulmonary pattern due to**

peribronchial and perivascular interstitial fibrosis. The pleura is thickened, deformed by basal adhesions.
At *stage II*, **general clinical manifestations of the disease are aggravated. Cyanosis increases. Disturbed by shortness of breath at the slightest load. Seizures of dry, unproductive cough are becoming more frequent, with a small amount of difficult-to-discharge, viscous sputum, accompanied by severe chest pains. Physical examination identifies signs of limited mobility of the lower edge of the lungs. The pleural friction noise, dry and wet rales are heard. On radiographs markedly pronounced consolidation, expansion, deformation of the roots of the lungs. Increased transparency of the pulmonary fields (emphysema). The pulmonary pattern is roughly cellular, sharply reinforced. There may be signs of dilatation of the right atrium and ventricle, expansion of the cone of the pulmonary artery (signs of pulmonary heart). The ECG shows signs of right atrial and ventricular hypertrophy, diffuse changes in the myocardium of both ventricles (myocardial dystrophy).**
Stage III asbestosis **is characterized by a severe general condition of patients. They are exhausted, cyanotic. They are worried about constant shortness of breath at rest, unproductive cough with attacks of breathlessness, pain in the chest. With decompensation of the pulmonary heart, the abdomen (ascites) increases, and peripheral edema appears. In connection with the development of severe emphysema, the chest becomes barrel-shaped. Non-sound crepitations (emphysema, pneumosclerosis) are tapped above the lungs.**
The diagnosis of asbestosis is justified by the following circumstances:
• Professional route confirming long-term work in industries related to the extraction, processing, use of asbestos in the production of the mineral.
• The results of the hygienic examination, indicating the excess of the maximum permissible concentrations of asbestos dust in the air at the workplace of the sick person.
• Typical clinical manifestations of the disease.
• Detection of asbestos fibers and bodies in sputum.
• The results of the study of respiratory function, X-ray, ECG, and other methods confirming that the patient has characteristic changes in the structure of asbestosis of the lungs, respiratory failure, signs of pulmonary heart.

Test 36. **Anthracosis - pneumoconiosis resulting from the inhalation of slightly fibrogenic dust containing mainly carbon particles (coal, graphite, soot). It is found in people working in the coal industry, in the production of graphite, coal electrodes, and by-product coking plants. Anthracosis is characterized by the accumulation of coal dust in the lung tissue, which causes the characteristic black color of the lung. In places of concentration of coal dust macrophages concentrate. As a result, anthracotic foci appear, which can merge into large fields of anthracotic fibrosa. In such foci, necrosis may occur with the formation of decomposition cavities - anthracotic cavities. The degree of pulmonary fibrosis in anthracosis is directly dependent on the presence of high fibrogenic silicon dioxide in impurities in carbon dust. In the presence of more than 10% silica in dust, another, less favorable pneumoconiosis - anthraco-silicosis occurs.**
Anthracosis usually develops after 10–12 years of constant work in conditions of high content of carbon dust in the ambient air.

Test 37. **Anthracosis refers to relatively favorable for the clinical course of pneumoconiosis. Its clinical picture is largely determined by comorbid diseases - chronic dust bronchitis, pulmonary emphysema. It is for this reason that the leading symptoms of anthracosis are shortness of breath, dry cough with dark, even black sputum. Objective examination reveals symptoms of pulmonary emphysema (percussion sound with a box shade), chronic obstructive bronchitis (hard breathing, dry rales).**

Test 38. **Metallodiosis-siderosis is a relatively benign disease. The clinical manifestations of this disease are mild. Complaints, objective symptoms of respiratory organs and respiratory failure are absent. In the sputum, iron particles can be detected. An x-ray examination of the lungs in patients with siderosis shows diffuse, not pronounced peribronchial and perivascular fibrosis, scattered throughout all the pulmonary fields, with distinct, uneven contours, radiopaque foci of accumulation of iron particles.**

Test 39. **Berylliosis is a toxic-allergic pneumoconiosis caused by an agent that does not have fibrogenic activity — dust or aerosols of the rare-earth metal of beryllium or its compounds.**

Test 40. **Beryllium is part of the gemstones (beryl, emerald-dy, aquamarine, etc.), is widely used in the form of alloys with other metals in industry. The amount of beryllium produced is constantly increasing. Accordingly, the number of people in contact with this metal, potentially capable of contracting berylliosis, is also growing.**
Beryllium most often enters the body through the respiratory system in the form of vapor, dust, aerosols. Less commonly, it can enter through the digestive tract, penetrate by diffusion through intact skin. About 50% of beryllium that has entered the body is excreted through the kidneys and intestines, 25% is de-understood in the tissues of the lungs, liver, and kidneys, and another 25% is fixed in the bones, replacing magnesium there. Beryllium is most firmly and permanently fixed in the bones and lung tissue.
Unlike other pneumoconiosis, berylliosis develops when breathing in minimum concentrations of aerosols containing metallic beryllium or its compounds. The maximum permissible concentration of beryllium in the surrounding air is 0.001 mg / m3. For the formation of berylliosis, even short contact with minimal concentrations of beryllium is enough even within a few minutes.

Tests 41, 42. **Intake of beryllium in the body leads to the formation of hypersensitive immune complex pneumonitis with increased levels of antibodies to DNA, RNA, various protein components of the lung tissue, bronchi, liver, followed by the development of diffuse granulomatous pneumofibrosis with marked respiratory failure.**
Beryllium is capable of forming colloidal precipitates, in which molecules of proteins and nucleoproteins change their structure so much that they are no longer recognized by the immune system as "their own". An autoimmune delayed-type hypersensitivity reaction occurs. In the lungs, in the places of fixation of beryllium in the wall of the bronchioles, the alveolar septa form foci of non-caseating granulomatosis, consisting of epithelioid cells. In the central part of the granulomas, shell-shaped (conchoidal) beryllium bodies are located. Microscopic granulomas merge into large granulomatous nodes. These nodes create a characteristic tuberosity of the surface of the lungs, and on the cut of the lungs they have the form of small grayish-white formations. Granulomas of the same structure can be found in other organs and tissues - the lymph nodes, skin, liver, spleen, kidneys.

Test 43. **The acute form of berylliosis when metal aerosols are delivered through the respiratory tract is manifested in a number of fairly characteristic syndromes:**
Acute tracheobronchitis. **Caused by the irritating effect of beryllium vapor and aerosol. Manifests dry cough, shortness of breath, chest pain, aggravated by coughing. Hyperemia of the mucous membranes of the upper respiratory tract is detected. Radiographically revealed increased pulmonary pattern.**
Acute broncho-bronchiolitis and / or toxic pneumonitis. **Characterized by severe general condition with fever, chills, shortness of breath, paroxysmal unproductive cough, torokalgiey. Auscultation in light crepitus, moist rales. Concurrent conjunctivitis, dermatitis, and skin erythema may occur simultaneously.**

Clinical manifestations of acute beryllium intoxication persist for 2-3 months. If a lethal outcome has not occurred in 2-3 weeks, the disease enters a resolution phase with subsequent recovery. Repeated acute berylliosis may occur upon repeated contact with beryllium.

Test 44. **The chronic form of berylliosis is formed 1-2 years after the moment of the first contact with this metal. The disease begins with shortness of breath, which progresses rapidly, becomes agonizing, accompanied by dry coughing, suffocation. Concerned about pain in the chest. Recurrent fever appears.**

Test 45. **An objective examination in the case of the presence of chronic berylliosis revealed diffuse warm cyanosis. The terminal phalanges of the fingers have the appearance of "drum sticks", the nails - "watch glasses".**
In the lungs, above all the fields, non-sound crepitus sounds are heard, similar to "cellophane crackling", dry and moist finely wheezing. Borders of the heart are expanded, tones are muffled. II tone above the pulmonary artery is accentuated.
With further development of the disease, there are signs of pulmonary heart decompensation (hepatomegaly, ascites, edema).

Test 46. **Radiologically isolated interstitial and granulomatous forms of berylliosis. At the initial stage of berylliosis, the vascular and broncho-pulmonary pattern is strengthened, deformed, which corresponds mainly to interstitial changes. With the further development of the disease, small dotted nodules (confluent foci of granulomatosis), compaction, expansion of the roots of the lungs appear.**

*Test 4*7**. Pneumoconioses from mixed dusts are among the most common. Depending on the amount of free silica contained in the mixed dust, its fibrogenic activity changes. The presence in the mixed dust of a large amount of silicon dioxide, toxic-allergenic components (beryllium) can significantly aggravate the severity of the clinical manifestations of the disease.**
The most common pneumoconiosis caused by mixed dust are: siderosilicosis, silicoanthracosis, electric welders pneumoconiosis.

Tests 48-50. **Electric welding is accompanied by the release into the air of a gas-aerosol mixture of complex composition. In this mixture there are microparticles of condensate evaporating from the melt at the place of welding: metals that make up the part to be welded and the electrodes and their oxides, flux components, including free silicon dioxide. As well as a large amount of aggressive gases - ozone, nitrogen oxides, carbon, hydrogen fluoride, etc. Inhalation of such a mixture causes irritation of the upper respiratory tract, leading to the formation of chronic bronchitis. Fixation of particles of metal and their oxides in the bronchi and alve-ola leads to pathological changes in the lungs, typical of poorly fibrogenic benign metallooniosis. Most often it is siderosis of the lungs with a clinically favorable course.**
The high content in welding gas-aerosol mixtures of highly fibrogenic silicon dioxide is the cause of more severe pulmonary pathology — siderosilicosis. Inhalation of welding products made with high chromium-containing electrodes can lead to the complication of pneumoconiosis by professional bronchial asthma.
The presence of beryllium in the parts being welded is one of the leading causes of toxic-allergic pneumonitis, diffuse granulomatous pneumofibrosis.
When casting and welding with non-ferrous metals in closed premises, workers may experience acute intoxication, accompanied by high body temperature - “casting fever”.

Test 51. **Byssinosis is a peculiar bronchospastic syndrome, resulting from the interaction of the bronchial mucosa with fibrous vegetable dust. In the first place with the dust of flax, hemp, less cotton, jute, sisal.**
Currently, byssinosis is excluded from the group of pneumoconiosis, arising from exposure to organic dust, as well as from the group of hypersensitive pneumonitis.

Test 52. **Unlike dust bronchitis, for the onset of byssinosis, a much smaller concentration of dust in the inhaled air is needed. The damaging effect of fibrous plant dust is due to the presence on the surface of fibers of bacteria, fungi that produce biologically active substances, including histamine-like substances. Cotton dust may contain components that inactivate the action of histaminase in the bronchial mucosa. Characteristic is the formation of sensitization to the components of fibrous dust, which is confirmed by the presence in the blood of patients with byssinosis of specific IgE antibodies to the proteins of the mold fungi, usually present on plant fibers. All these reasons contribute to the formation of the main syndrome of byssinosis - bronchospastic.**

Test 53. **Dusty damage to the lungs in byssinosis has a clinical picture that distinguishes this disease from chronic dust bronchitis. There are three stages of the disease. Most often I stage. Byssinosis of the second and, especially, the third stage is rarely formed.**
Stage I **byssinosis is characterized by the appearance of bronchospasm in the form of respiratory discomfort, a feeling of heaviness in the chest, and a dry cough that occurs several hours after starting work in dusty conditions. At the end of work and returning home well-being is restored. The most severe attacks, accompanied by suffocation, occur after the weekend - Monday syndrome. During the working week, the intensity of bronchospastic manifestations gradually diminishes. During the period of deterioration of health in patients with auscultation in the lungs dry whistling rales are heard. Attacks of bronchospasm may be accompanied by sub-febrile.**
Stage II **of the disease is manifested by longer periods of bronchospasm that do not go on throughout the week. There is shortness of breath, increasing attacks of suffocation, occurring not only on Monday, but also on other working days. Choking can provoke changes in meteorological conditions, physical stress. On weekends, patients' state of mind does not fully normalize. A dry, unproductive cough, chest tightness is preserved. In the lungs, against the background of harsh breathing, dry whistling rattles, often heard from a distance, are constantly heard.**
Stage III **byssinosis is a transition of the disease into a severe form of obstructive bronchitis or into a professional bronchial asthma. Pathological process in the lungs is characterized by severe ventilation disorders, the formation of chronic pulmonary heart.**

Test 54. **Chronic dust (professional) bronchitis - chronic diffuse non-allergic inflammatory disease of the bronchi, resulting from prolonged work in conditions of high content in the inhaled air of moderately aggressive mixed dust, leading to progressive violation of pulmonary ventilation and gas exchange on obstructive - type, manifested by cough, shortness of breath, sputum. May precede or be combined with occupational bronchial asthma.**

Test 55. **In chronic dusty professional bronchitis, endobronchitis occurs and gradually progresses, which can lead to irreversible obstruction of the bronchi. Endobronchitis is usually combined with nonspecific hyperreactivity of the bronchial muscles, which manifests itself as a bronchospastic response in response to exposure to dust factor.**

Test 56. **The period specific for chronic dust bronchitis is only the initial period of time corresponding to the first months of contact with the dust factor. This period is sometimes**

referred to as "bronchitis irritation." The further clinical course, objective, instrumental, radiological dynamics of chronic occupational dust bronchitis differs little from chronic bronchitis unrelated to the effects of occupational hazards. To form a comprehensive clinical picture of occupational dust bronchitis, at least 5-10 years of work in dusty inhaled air is required.
In its development, chronic bronchitis passes through three stages, corresponding to three degrees of severity of the clinical course of the disease.
Stage I - chronic mild bronchitis. **Corresponds to the primary lesion of the mucous membrane of the large bronchi (endobronchitis irritation). Accompanied by unproductive cough, mainly in the morning, with the separation of a small amount of mucous sputum.**

For persons working with industrial pollutants, stage I chronic dust bronchitis can last for 2 to 5 years. In such cases, there are periods of acute coughing with a total duration of up to 3 months per year for 2 years or more.
At auscultation on the I stage of professional dust bronchitis you listen to single dry buzzing, wheezing rattles that are not heard from a distance. According to x-ray studies, pathological changes can not be detected. In the study of respiratory function, the minimally expressed signs of non-obstructive pulmonary insufficiency (DN 0 - I degree) are rarely detected, mainly due to the formation in some patients of minor restrictive (difficulty in lung inhalation) changes due to the formation of earth of the lungs.

Test 57. **Stage** *II chronic occupational dust bronchitis* **- chronic obstructive bronchitis of moderate severity. It is characterized by severe disorders of the mucociliary apparatus of the bronchi, excessive secretion, and infection of the bronchial secretion. The inflammatory process penetrates deep into the walls of the bronchi. The production of a surfactant surfactant is impaired, which leads to the formation of pulmonary emphysema.**
At this stage, patients have a cough with a small separation of mucopurulent sputum almost throughout the year. There is a constant mixed inspiratory and expiratory dyspnea with a predominance of difficulty in exhaling.
When percussion over the lower parts of the lungs is determined by the "box" shade of the pulmonary sound, auscultatory - weakened vesicular breathing with an extended exhalation, dry wheezing.
In the study of respiratory function, you can install the DN I - II degree of obstructive or mixed type.
X-ray reveals increased transparency of the lung tissue, increased bronchial pattern, signs of moderate dilatation of the right lary heart and auricle.
The ECG shows signs of myocardial hypertrophy of the right atrial and ventricle.

Test 58. Stage III - severe chronic obstructive bronchitis **with severe restrictive obstructive pulmonary insufficiency, compensated or decompensated pulmonary heart. It corresponds to the development of intramural (affected all layers of the wall) of deforming bronchitis, sclerotic changes, leading to obliteration of the lumen of the small bronchi with the formation of multiple lung microatelectasis. It is these changes that lead to a disturbance of gas exchange in the lungs and impede blood flow through the pulmonary vessels.**
This stage is characterized by complaints of constantly disturbing cough with thick, difficult to separate, purulent sputum. Inspiratory dyspnea at rest is replaced by expiratory or mixed with physical activity of the patient. Coughing episodes may turn into asphyxiation. Appear cyanosis, pain in the right hypochondrium, hepatomegaly, peripheral edema, indicating decompensation of chronic pulmonary heart.
Respiratory function is significantly impaired, corresponds to DN II-III degree of mixed obstructive-restrictive type.

Hypoxemia is determined in the blood. The formation of compensatory erythrocytosis is possible.
On radiographs pronounced changes in the pulmonary pattern, signs of deforming bronchitis, diffuse pulmonary fibrosis, pulmonary emphysema, bronchiectasis. During the exacerbation period, foci of pneumonia can be detected, on the ECG - diffuse changes in the myocardium of all ventricles (myo-cardio dystrophy), hypertrophy of the right atrial myocardium and ventricle.

Test 59. **Occupational bronchial asthma is a disease commonly characterized by chronic inflammation of the airways that occurs in response to contact with certain chemicals in production. Bronchial asthma is determined by a history of symptoms from the respiratory organs, such as wheezing, shortness of breath, a feeling of congestion in the chest and cough, the severity of which changes with time, as well as the variability of limiting the speed of air flow during exhalation (Global GINA- 2014-2017).**

Test 60. **In industrialized countries, asthma is one of the most common forms of occupational lung disease. In 5-15% of the adult population suffering from bronchial asthma, the disease is caused by circumstances related to their professional activity. There are at least 240 factors of the working environment that can cause bronchial asthma. They are divided into two groups.**
***1. High molecular weight substances (molecular weight over 500 daltons*). These include:**
• allergens of animal origin (wool, down, epidermis, animal, bird, insect excreta);
• allergens of plant origin (dust of tobacco, jute, paper, wood, cotton, flax, natural silk, grain, hops);
• allergens of protein and enzyme preparations used in industry (rennet, trypsin, pancreatin, bromelin, papain, fungal amylase, yeast protein, etc.);
• seafood allergens (chitin shrimp, crabs, etc.).
2. Low molecular weight substances (molecular weight less than 500 daltons):
• diisocyanates used in the production of polyurethane and its derivatives;
• anhydrides used as hardeners in the manufacture of products from epoxy resin;
• metals - platinum, chromium, nickel (metallurgical production and processing of metals);
• fluxes used when soldering metals with light or refractory solders, aluminum soldering;
• some drugs and their ingredients;
• other low molecular weight chemicals with allergenic properties.

Test 61. **A high molecular weight substance alone or a low molecular weight substance (hapten), after conjugation with human proteins, causes an immune response in the form of hypersensitivity type I (atopic). Allergic non-IgE-dependent bronchial asthma due to IgG antibodies and / or a delayed-type hypersensitivity reaction (T-cells) may develop. Subsequent inhalation of high- or low-molecular substances at the workplace causes** *a rapid bronchospastic reaction* **that occurs a few minutes after contact with them or** *a delayed reaction* **that occurs after 4-6 hours, with a peak after 8-10 hours, and a decrease in reaction after 24-48 hours There may be a biphasic immune response, manifested as a quick, and delayed response. With an** *isolated late-life* **(often caused by agents of low molecular weight) - symptoms of asthma are observed at the end of the work shift, or even after it.**
With a massive, acute impact on the respiratory organs of an irritant, typical symptoms of bronchial asthma can appear in the form of expiratory dyspnea, dry wheezing, unproductive dry cough - *a syndrome of reactive dysfunction of the respiratory tract, corresponding to acute irritant asthma*. **Late asthmatic reactions that occur after acute massive exposure to irritants are due to the formation of nonspecific bronchial**

hyperreactivity. In such cases, the bronchial tree reacts with a spasm to many subliminal stimuli (cooling, exercise, dust, tobacco smoke) lasting up to 3 months.
Subacute irritant bronchial asthma **develops after several exposures of a high / moderate level stimulus. A worker may develop bronchial hyperreactivity, as in the case of acute irradiation asthma. An increase in the symptoms of the disease will occur after inhaling low doses of irritants, exercise, etc.**

Test 62. **For occupational bronchial asthma, the same drugs and principles of pharmacotherapy are used as with other forms of asthma. For attacks of breath, use the following medications:**
• short-acting β2-agonists (salbutamol, fenoterol);
• β2-agonists of long-acting with a rapid onset of action (formoterol) - only for patients receiving inhaled glucocorticosteroids *(inhaled corticosteroids);*
• fixed combinations of inhaled corticosteroids (budesonide, beclomethasone, mometasone, fluticasone propionate) + formoterol is the "single inhaler mode", which is used both to relieve asthma symptoms and for basic therapy;
• inhaled short-acting anticholinergics (ipratropium bromide, troventol) - 2nd line drugs (shown in case of insufficient response to short-acting β2-agonists);
• combined bronchodilators, including short-acting β2-agonists and inhaled anticholinergics: fenoterol + ipratropium bromide (berodual); salbutamol + ipratropium bromide;
• short-acting theophylline (intravenous) - aminophylline, aminophylline. Used with severe exacerbation of asthma and the presence of refractoriness to β2-agonists and M-cholinolytics, in the absence of inhaled β2-short-acting agonists or their delivery vehicles (nebulizer), or in case of refusal from inhalation therapy. A weaker bronchodilator effect, use associated with frequent side effects (nausea, vomiting, tachycardia, rhythm disturbances, psychomotor agitation);
• systemic corticosteroids (IV, oral). Used in asthma attacks, if the appointment of β2-agonists of short-acting did not lead to improvement.

Test 63. **Step therapy of occupational bronchial asthma is carried out according to the same rules as asthma, which is not associated with production factors (table).**

Table - Step approach to control symptoms and minimize future risks of bronchial asthma (adapted from GINA-2014-2017)

Preferred choice of control therapy	**Step 1**	**Step 2: low doses of iGX**	**Step 3: low doses of inhaled corticosteroids / DBA; SLIT§**	**Step 4: Medium / high doses of inhaled corticosteroids; SLIT**	**Step 5: Assign adjuvant therapy: anti-IgE or anti-IL-5**
Other Controlling Options	**Consider low doses of inhaled corticosteroids ***	**ALT or low doses of theophylline ***	**Medium / high doses of ICS; or low doses of IGX + ALT; or IGX + theophylline***	**Add Tiotropium Bromide # High doses of inhaled corticosteroids + ALT; or iGX + theophylline***	**Add low doses of oral GCS**

Relief of asthma symptoms short-acting	**β2 agonists by need Short-acting**	**β2-agonists or low doses of inhaled corticosteroids / formoterol ** according to need**
Remember the importance of: patient education (action plan, self-control, regular check-ups); treatment of associated diseases (obesity, rhinosinusitis, food allergies, etc.); eliminate the risk factors for asthma exacerbations (uncontrolled symptoms, use of> 1 β2-agonist inhaler 200 doses / month, inappropriate inhalation technique, smoking, contact with allergens); non-pharmacological therapy (physical activity, weight loss, smoking cessation). *Consider going up* **a step if asthma symptoms are not controlled, there are risks of exacerbations and adverse outcomes, but first check the correctness of the diagnosis, inhalation technique, evaluate adherence to therapy.** *Consider going down* **a step if symptoms are controlled for 3 months, low risk of exacerbations. Discontinuation of ICS treatment is not recommended.**		
Notes: 1. ALT - leukotriene receptor antagonists; DBA - long acting β2-agonists; anti-IL-5 - antibodies against interleukin-5; **2. * - for children 6-11 years old, theophylline is not recommended, the preferred choice is the average dose of inhaled corticosteroids;** **3. ** - for patients who receive low doses of budesonide / formoterol or beclomethasone / formoterol, it is recommended to use low doses of inhaled corticosteroids / formoterol for the treatment of asthma symptoms (a single inhaler for maintenance therapy and relief of symptoms).** **4. # - Tiotropium bromide has been used since the age of 18.** **5. § - SLIT - sublingual allergen-specific immunotherapy in case of sensitization to house dust mite, if there are aggravations against inhaled corticosteroids (performed with FEV1> 70% of the proper indicator).**		

To maintain remission (basic therapy) of patients with occupational bronchial asthma, apply:

• inhaled glucocorticosteroids;

• leukotriene receptor antagonists;

• long-acting β2-agonists and combined forms: inhaled corticosteroids + β2-short-acting agonists; inhaled corticosteroids + β2 long-acting agonists;

• long-acting anticholinergic drugs (tiotropium bromide) and fixed combinations: anticholinergic drugs + short-acting β2-agonists;

• inhaled forms of cromoglicic acid or nedocromil sodium;

• prolonged action theophyllines;

• systemic glucocorticosteroids;

• biological drugs (anti-IgE, anti-interleukin-5).

A stepwise approach to treatment must be accompanied by the elimination of allergens from the patient's environment, and effective control over triggers. In patients with occupational bronchial asthma, this is accomplished by rational employment of the patient outside of contact with industrial allergens.

EXPLANATION TEXTS
to control tests section III
"PROFESSIONAL DISEASES CAUSED BY EXPOSURE TO PHYSICAL FACTORS"

Tests 1-6. **Vibration disease is a professional pathology caused by the prolonged effect of mechanical vibrations on the human body under production conditions.**
The whole body or its individual parts can be subjected to vibration of pathological intensity and duration by people working with manual pneumatic and electric tools - jackhammers, perforators, etc., while controlling machines that have in their design return and translational and quickly spinning unbalanced mechanisms - forge-pressing equipment, crushing plants, etc., when working in the cabins of vehicles - helicopters, airplanes, tractors, etc. Vibration disease occurs if steam The meters of vibration impact on the worker exceed the maximum permissible sanitary and hygienic standards.
Vibration can be exposed to the whole body. This is the vibration of the general effect on the whole organism Such vibration can occur in drivers of vehicles, in workers engaged in vibrocondensing of concrete, etc. If vibration affects certain areas of the body — usually the arms — this is a local vibration. Most often it happens to work with a jackhammer, riveters, blacksmiths. Perhaps the combined effects of local and general vibration - mixed vibration.
The concept of vibration as a pathogenetic factor capable of causing disease includes the following points:
Vibration - the oscillatory movement of the body in the form of a periodic, pendulum-shaped deviation from the position of stable equilibrium.
The frequency of vibrations is measured by the number of deviations in both directions from the equilibrium position per unit of time. One oscillatory motion in 1 second is designated as 1 Hz. Oscillations with a frequency not exceeding 16 Hz are called infrasonic. They are felt tactilely, but not perceived by an auditory analyzer. Fluctuations from 16 to 20,000 Hz are called sound. They are perceived by ear. Oscillations above 20,000 Hz are ultrasonic. Human analyzers do not perceive them.
The magnitude of the maximum deviation from the equilibrium position is called the amplitude of vibration and is measured in linear metric units - mm, cm.
The vibration velocity (m / s) and vibration acceleration (m / s2) are derived from the frequency and amplitude of vibrations. The threshold of onset of sensations of vibration is 10–4 m / s, the threshold of onset of pain is 1 m / s.
The strength (energy) of vibration is directly proportional to the frequency and amplitude of oscillations. It is measured in decibels (dB).
Vibration, as a factor in the pathological effect on humans, is divided into three frequency ranges — low frequency, mid-frequency and high frequency. From the hygienic point of view, the frequency ranges differ depending on whether this is a general or local vibration.
• Under general exposure, the low-frequency range corresponds to 1–4 Hz, the mid-frequency range is 8–16 Hz, and the high-frequency range is 31.5–63 Hz.
• For local vibration exposure, the frequency ranges are shifted up: 8–16 Hz — low frequency, 31.5–63 Hz — mid-frequency, 125–1000 Hz — high frequency.
For mechanical objects subject to vibration, there is the concept of resonance - the frequency with which this object can perform free oscillations like a pendulum. The resonant frequency corresponds to the maximum amplitude of the object's vibrations. The human body has a resonance at a frequency of about 6 Hz, the head of an average person - about 8 Hz. Vibrations on such frequencies extremely negatively affect the general state of health and the activity of the central nervous system.
The zone is affected by high-frequency vibrations less than low-frequency ones, since with an increase in the frequency of vibrations, the intensity of their absorption along the propagation path increases.

Tests 7-11. **High intensity vibration with vibration velocity greater than 1 m / s is a traumatic factor. As a result of its action, injuries can occur up to tearing of the skin, muscles, and internal organs. Vibration of lower intensity with prolonged exposure causes impaired neurohumoral regulation of vascular and muscle tone. Under its action, receptor structures, nerve endings in the system of perception and control of pain, tactile sensations are dulled or even atrophy. There are pathological changes in the centers of regulation of pain and tactile sensitivity. This leads to a variety of neurovegetative and dystrophic changes in the internal organs.**

The main conductor of vibration oscillations in the human body are the bones of the skeleton. The vibration energy is extinguished in the ligaments, cartilage structures, causing dystrophic changes in the joints of the extremities (osteoarthritis), spinal column (osteochondrosis, spondyloarthritis).

Vibration in the range of 100-250 Hz affects the rheological properties of blood, reducing its characteristic viscosity. Therefore, under the influence of vibration at the same width of the vessel, the volumetric rate of blood flow in it increases. In order to preserve adequate blood circulation, compensatory activation of the vasoconstrictor mechanisms occurs in the tissue undergoing vibration, which balances the hemodynamic effects of vibration. With the elimination of vibration equilibrium is disturbed. The activated mechanisms of vasoconstriction in the absence of vibration lead to a decrease in blood flow, ischemia of parts of the body that were previously subjected to vibration. It is for this reason that patients with vibration disease suffer from local effects on the hands of the syndrome of "cancellation of vibration." Outside of working with a vibrating instrument, they suffer from ischemic pain in their hands. Having come to work and once again taking up a vibrating instrument, they no longer feel these pains, since the blood circulation of their limbs is restored. The same sensations occur in the legs in patients with vibration disease from the overall impact.

Test 12. **Distinguish vibration disease from the effects of local vibration, vibration disease from the effects of general vibration and vibration disease from the effects of mixed (local and general) vibration.**

Vibration disease from the effects of local vibration most often occurs in persons working with a manual vibrating tool (jackhammers, perforators, saws, etc.). The disease is forming very slowly. Persons with a long history of work with vibrating tools suffer from it.

Test 13-14. **Angiodystonic syndrome is characteristic of vibratory disease from local exposure - those who work with vibrotools experience numbness of fingers and hands at rest, especially at night, on weekends. The hands become very sensitive to even a little overcooling, then become pale, cold, insensitive to tactile and painful stimuli, and after a while - purple cyanotic, pasty. There are severe pains in the fingers. Outside of working with a vibrating instrument, aching pain in knees and forearms may disturb. With the start of work in terms of vibration, all these sensations quickly disappear. In the later stages of the disease, angiodistonic shifts can turn into acroasphyxia with necrosis of the terminal phalanges of the fingers.**

Along with angiodystonic syndrome, autonomic-sensory polyneuropathy usually occurs. It is characterized by impaired peripheral vibration, pain, and temperature sensitivity of the polyneuritic type, trophic disorders. Tactile and muscular-articular sensitivity usually do not change.

In the first place, vibration sensitivity is dulled. The severity of the changes is proportional to the severity of the vibration disease. A rough estimate of the vibration sensitivity can be made by measuring the duration of sensations of the gradually decreasing vibration of the tuning fork. Accurate assessment is performed by a special device - palletestiometer.

Pain sensitivity is greatly affected. At the very beginning of the disease, there is a short period of heightened pain sensitivity — hyperegestion, but then hypostezia appears and gradually worsens. First, the painful sensitivity of the fingers, especially of the hands and forearms, is weakened. If the legs were subjected to local vibration, similar changes in pain sensitivity occur on the fingers, feet, legs.
Trophic disorders are manifested by hyperkeratosis of the palmar surface of the hands, side surfaces of the fingers. Often there is a focal hyperkeratosis - pachidermy in the form of rounded, smooth, pale formations on the outer surface of the interphalangeal joints. Multiple cracks in the skin of the fingers, deformation and thinning of the nails appear.
The defeat of the musculoskeletal system is manifested by myositis with affection of the muscles of the shoulder girdle, myofasciculitis, and forelimb tendenzomiosis. There are spinal lesions in the form of osteochondrosis, spondyloarosis. Osteoarthritis is formed with a lesion of the elbow and shoulder joints. With prolonged exposure to intense local vibration, aseptic necrosis of the humeral head can occur.
Since the vibration is usually accompanied by noise, patients may experience quite severe damage to the hearing aid. Most often formed bilateral acoustic neuritis of the auditory nerve, with hearing impairments, diagnosed by audiometry.
Somatic disorders appear. First of all, these are functional disorders of the central nervous system, manifested by nevrozopodobnoy symptoms. Sometimes functional diseases of the stomach, intestines, dyskinesia of the biliary system are formed. In advanced stages of the disease, dyscirculatory encephalopathy often occurs, usually in the form of encephalopolyneuropathy.

Tests 15-17. **Vibration disease from local exposure to vibration can have the following three degrees.**
The first degree **of severity of vibration disease. Fully reversible phase of the disease. Clinical manifestations are minimal, are functional in nature:**
• Peripheral angiodystonic syndrome of the upper extremities with rare angiospasms of the fingers.
• Syndrome of autonomic-sensory polyneuropathy of the upper extremities.
Second degree **It is characterized by the formation of persistent neurohumoral disorders with a developed clinical picture of the disease in the form of the following syndromes:**
• Peripheral angiodystonic syndrome of the upper extremities with frequent angiospasms of the fingers.
• Syndrome of autonomic-sensory polyneuropathy of the upper extremities:
o with persistent vegetative-trophic disorders on the hands;
o with dystrophic disorders of the musculoskeletal system of the arms and shoulder girdle (myopatosis, myofibrosis, periarthrosis, arthrosis); with neck and shoulder plexopathy;
o with cerebral angiodystonic syndrome.
*The third degree of severit***y. Corresponds to the appearance of gross, irreversible anatomical disorders with clinical manifestations in the form of the following syndromes:**
• Syndrome of sensory-motor polyneuropathy of the upper extremities.
• Encephalopolyneuropathy syndrome.
• Syndrome of polyneuropathy with generalized acroangiospasms.

Test 18. **Vibration sickness from exposure to a general vibration occurs in people working under conditions of a general vibration of the workplace - drivers of vehicles, helicopter pilots, etc.**
Vegeto-vestibular syndrome is the leading clinical manifestation of this form of the disease. Manifested with periodic dizziness, migraine-like bouts of intense headaches. During dizziness, hearing and vision may be impaired. In the interictal period, many patients continue to be disturbed by headaches, increased irritability.

There is a syndrome of autonomic-sensory polyneuropathy of the extremities. Characterized by cyanosis, cooling, pastoznost, sweating hands. It is manifested by paresthesias, pains in the arms and legs more at night, on high days. Patients may experience atrophy of the proximal muscles on the legs. Raises the threshold of vibration and pain sensitivity.
Damage to the central nervous system is manifested by vegetative-dystonic disorders, neurasthenia.
Multiple functional disorders of the internal organs are formed. Occur functional diseases of the stomach, intestines, discs of the biliary system. In women, general vibration can cause dysmenorrhea.

Tests 19-21. **Vibration disease from exposure to general vibration has three degrees of clinical manifestation.**
The first degree of severity **(initial manifestations) corresponds to the stage of neuro-reflex disorders. The clinical picture of the disease at this stage is formed by:**
• Central or peripheral angiodystonic syndrome.
• Vegetative vestibular syndrome.
• Syndrome of sensory or autonomic-sensory polyneuropathy of the lower extremities.
The second degree of severity **(moderately pronounced manifestations) corresponds to the stage of neuro-humoral disorders. Its components are:**
• Cerebral-peripheral angiodystonic syndrome.
• Syndrome of sensory or autonomic-sensory polyneuropathy in combination:
o with polyradiculo-neuropathy syndrome;
o with secondary lumbosacral radicular syndrome due to osteochondrosis of the lumbar spine;
o with functional disorders of the nervous system - neuro-sthenia syndrome.
The third degree of severity **corresponds to the stage of irreversible structural changes in the nervous system, bones, muscles, joints. The leading clinical manifestations of this stage of the disease are:**
• Syndrome of a touch and motor polyneuropathy.
• Dyscirculatory encephalopathy syndrome combined with peripheral polyneuropathy - encephalopolyneuropathy syndrome.
• Common osteoarthritis of the spine, large joints of the end.

Test 22. **Due to the fact that the clinical symptoms of vibration disease are not specific, especially in the initial stages of the development of the disease, the diagnosis of this pathology should be carried out using clinical and physiological methods:**
• To assess the state of peripheral circulation are used:
- **Skin thermometry using an electronic point thermometer in symmetrical areas of the arms and legs with the determination of the time it takes for the temperature to recover after a cold test.**
- **Thermoscopy using a thermal imager with a cold projection.**
- **Reovasography of the peripheral vascular bed and occlusive plethysmography of the extremities using impedance reopletism graph.**
- **Optical capillaroscopy of the vessels of the nail bed, conjunctiva.**

• To identify disorders (sensitive) of the peripheral nervous system, apply:
- **Algesimetry - reveals the severity of pain sensitivity disorders using an algesimeter.**
- **Palletestiometry - reveals, with the help of a tuning fork or pallets-ziometer, violations of the vibration sensitivity threshold.**

• To identify violations of the neuromotor apparatus are used:
- **Stimulation electroneuromyography.**

- **Needle electromyography with a study of the rate of propagation of excitation in motor and sensory nerve trunks.**

Test 23. **The diagnosis of vibration disease is established upon receipt of a combination of the following data:**
• Professional route, indicating a long stay in a production environment characterized by local or general exposure to vibration.
• Sanitary and hygienic characteristics of the workplace of the diseased with an indication of the type and level of intensity of vibration exposure.
• Identification of clinical syndromes typical of vibration disease.
• Objective instrumental recording of signs of vibratory disease (from local vibrations), which include: changes in microcirculation, reduced vibration and pain sensitivity, slower recovery of skin temperature after cold test, changes in the bioelectric activity of muscles, slow down - excitation distress along the fibers of the motor nerves, violation of the complex electrical resistance of the skin. The most frequent signs of the impact of the overall vibration are changes in the function of the vestibular analyzer, polyneuropathy.
• Detection of X-ray changes in the state of the osteo-articular apparatus (osteoarthritis) arising from vibration disease.
• Registration of changes in the cardiovascular system according to ECG, echocardiography, Doppler echocardiography, characteristic of vibration disease.

Test 24. **Noise is one of the most common adverse factors of professional activity. It occurs when various mechanisms operate, having in their construction elements that vibrate with a frequency that corresponds to the range of human hearing - from 16 to 20,000 Hz.**
In contrast to the vibration effect, which is directly transmitted to the human body from a vibrating solid, the noise effect is transmitted from the source to the body working through the air in the form of acoustic oscillations of various amplitudes and frequencies.
Under conditions of intense noise, work of riveters, mechanics for the maintenance of jet engines, operators of crushing installations, power-saw benches, etc., takes place.
Characteristics of noise are frequency spectrum and intensity. Noises do not have a strict oscillation frequency, but are a mixture of sounds of different pitch and intensity with a predominance of certain frequencies. Depending on the dominant frequency spectrum, low-frequency, mid-frequency and high-frequency noises are distinguished. The maximum permissible level of noise exposure to a person in the workplace is 80 dB.

Test 25. **Acoustic noise fluctuations primarily affect the listening apparatus. The human ear is especially sensitive to sound vibrations in the frequency range of 1000-3000 Hz. Acoustic effects with an intensity of more than 80 dB can cause pain.**

Test 26. **Noise with an intensity of more than 120 dB is able to destroy the membrane and the sound-transmitting structure of the inner ear. In near explosions, a pulse of superintense noise arises, the impact of which causes barotrauma with temporary hearing loss. Such barotrauma often causes destruction of the eardrum and instantaneous death of the spiral organ, leading to complete hearing loss.**
With prolonged and intense exposure to acoustic oscillations, osteoarthritis of the auditory bones can occur. The result is a violation of sound conduction with the formation of a hearing loss syndrome. Intense vibrations of the bones of the skull can be transmitted and affect the human hearing aid, bypassing the airway of the auditory canal and the eardrum. As a result of the double impact of noise on the hearing aid through the ear canal and through bone conduction, dystrophic changes occur in the cochlear apparatus. Constant intense irritation of the auditory nerve endings leads to bilateral neuritis of the auditory nerve, which further increases hearing loss. Initially, the perception of sound vibrations

with a frequency higher than 4000 Hz is disturbed, then the threshold of perception of lower frequency oscillations, including conversational speech, corresponding to the range of 400-2000 Hz, rises.
Intense noise can directly affect an unprotected human body, causing it to vibrate. Pathological changes from such an influence of noise is no different from vibration disease from the general impact. They can result in the formation of vegeto-vestibular syndrome.
The combined acoustic effect on the hearing aid and the noise caused by the vibration of the body of the worker form disturbances of the central nervous system in the form of neurocirculatory dystonia.

Tests 27-30. **Local noise pathology in the form of a sensorineural ear-ear is formed after 15-20 years of work experience in the conditions of noise exceeding sanitary standards. Initially, diseases cease to be perceived as high sounds in the range of 4000-8000 Hz, then medium ones - 400-2000 Hz, and lastly - low frequencies - 16-100 Hz.**
Along with hearing loss, there are complaints of irritability, unstable headaches, dizziness, symptoms of neurocirculatory disorder, arterial hypertension. For some persons working under conditions of intense noise, these complaints may occur long before the appearance of symptoms of hearing loss.
According to the degree of hearing impairment, according to audiometry, the following degrees of occupational hearing loss are distinguished from working in noise conditions (table).
• I degree. Corresponds to minor changes in sound perception. A hearing loss of 1-10 dB is observed.
• II degree. Corresponds to the occurrence of neurosensory hearing loss with a slight decrease in hearing by 11–20 dB.
• III degree. Corresponds to the formation of sensorineural hearing loss with moderate hearing loss of 21-30 dB.
• IV degree. Corresponds to the appearance of sensorineural hearing loss with pronounced decrease in hearing by 31-45 dB.

Table - Degrees of occupational hearing loss from working in noise

Degree of hearing loss	Total threshold audiometry (dB)		The perception of the neck of sweat (m)
	Hearing loss for sound frequencies — you are 500, 1000 and 2000 Hz	Hearing loss of 4,000 Hz and the range of possible fluctuations	
Signs of noise exposure to the organ of hearing	to 10	50±20	5±1
Cochlear neuritis with mild hearing loss	11-20	60±20	4±1
Cochlear neuritis with a moderate degree of hearing loss	21-30	65±20	2±1
Cochlear neuritis with a significant	31-45	70±20	1±0,5

degree of hearing loss			

Test 31. **Diagnosis of lesions caused by prolonged work in conditions of intense noise is based on the following criteria:**

• Professional route confirming the fact of working in conditions of noise exceeding 80 dB for at least 10 years.

• The results of sanitary-hygienic examination of the workplace of the sick person with an assessment of the nature and intensity of noise exposure.

• Clinical manifestations of hearing loss in combination with functional changes in the central nervous system.

• Results of clinical and audiological studies confirming one or another degree of hearing loss.

• Long-term preservation of 100% speech intelligibility.

• Bilateral lesion of the organ of hearing.

• Other, except professional, causes of ear and ear (chronic otitis media, toxic damage to the hearing organs, etc.) are excluded.

EXPLANATION TEXTS
to control tests section IV
“PROFESSIONAL DISEASES CAUSED BY RE-VOLTAGE OF SEPARATE ORGANS AND SYSTEMS

Test 1. **Prerequisites for the occurrence of occupational diseases from the overvoltage of individual organs and systems are:**
• excess of hygienic standards of the number of stereotypical labor movements - over 8,640 per shift for local, non-work-related personal electronic computers, over 40,000 per shift - when working on personal electronic computers, and over 6,000 movements - for regional loads with the force up to 10% of the maximum arbitrary force;
• excess weight of lifted and transported cargo - single more than 10 kg for women and more than 30 kg and for men, the total mass of the transferred cargo during each hour of changing from the working surface is over 350 kg for women and over 870 kg for men;
• static load on the hands (pressure, holding on weight) of more than 42,000 kg / s for women and over 70,000 kg / s for men;
• prolonged stay in a forced, uncomfortable position whiter than 35% of the time during a work shift.
In the International Classification of Diseases 10 revision, the professional diseases of the musculoskeletal system caused by overstrain are included in section M70 "Diseases of soft tissues associated with exercise, overload and pressure." Muscle diseases that occur during functional overstrain are included in section M.70.8. Osteoarthritis, which is formed in the joints due to overloads, is encrypted under the rubrics M.18.1 and M.19.1.
Occupational diseases from functional overvoltage can occur in persons whose labor process is associated with:
• with lifting, holding on weight, moving heavy and oversized objects;
• with work in conditions of forced, non-physiological posture with excessive flexion, extension, rotation, displacement of the center of gravity of the body;
• with stereotyped, repeatedly repeated movements.

Test 2. **Myofibrosis is one of the most common occupational diseases of striated muscles. It occurs in the muscles that experience the greatest stress in the process of performing production operations. First of all, the muscles that provide stereotyped movements, maintaining a forced posture, keeping massive objects on weight are affected.**

Test 3. **Constant muscle tension is accompanied by compression of the feeding arteries, which leads to chronic ischemia, necrobiotic processes, and activation of fibrogenesis. As a result of chronic ischemia of the nerve endings and receptors, multiple patches of muscle hyper-irritability are formed in the form of painful trigger points. Most of these checks can be clinically latent, painless. But with cooling, additional muscle ischemia during their long-term contraction, such points can be activated, causing pain, neuromuscular dysfunction, manifested by spontaneous contractions of individual groups of muscle fibers. Active ischemic fibrogenesis leads to compaction of the muscles, reducing their contractility.**
With local static and dynamic loads, professional myofibrosis develops in the long and short wrist extensors, round pronator, brachiocephalus, radial and ulnar flexors of the wrist. When regional static-dynamic loads affect the muscles of the upper shoulder girdle - two-headed, deltoid, brachial. With the forced posture of the body, head, combined with excessive rotation, bending, lifting and holding weights, professional myofibrosis of the neck and lumbar muscles develops.

Test 4. **The initial period of professional myofibrosis is characterized by complaints from patients about intermittent pain, discomfort, and a feeling of tightness in muscles exposed to intense overload.**
In the future, pains begin to occur during motor activity, especially at the beginning of the movement, are evaluated by patients as deep, dull. A painful protective spasm that arises during movement blocks the extension of the extensors while reducing the flexors and vice versa. For this reason, the strength and amplitude of muscle contractions are reduced.
In the developed stage of the disease, the constant tension of the affected muscles is determined, and acutely painful trigger points are palpated.
Continued work under conditions of intense muscular load leads to a pathological increase in muscle density, which becomes gamenous to the touch. Increased pain, cramps that limit the amount of muscle movement. Against this background, the muscles begin to decrease in volume, lose their tone, become flabby. Muscular strength progressively weakens. Patients are unable to perform work that requires mouse effort.

Test 5. **For the diagnosis of occupational myofibrosis, the following criteria are used:**
• Professional route indicating the duration of work in conditions of overload of the muscular system.
• Assessment of the physical labor of the patient in accordance with hygienic criteria.
• Clinical symptoms of muscle damage.
• Detection in the blood of working patients with elevated levels of myoglobin, high activity of creatine phosphokinase.
• Exclusion of other diseases that cause muscle damage, muscle weakness is not related to professional activity (dermatomyositis-polymyositis, chronic adrenal insufficiency, etc.).

Test 6. **Professionally-related tendovaginitis, tendonitis, and ten-dosinovitis most often develop in the distal tendon parts of the forearm extensors. Less commonly, tendons and tendon sheaths of the biceps muscles, short shoulder rotators are affected.**

Test 7. **Professional tendovaginitis (tendinitis, tendosynovity) are formed under conditions of prolonged physical overstrain with static loads that occur when weights are held on the weight, as well as during dynamic overloads associated with fast movements of objects. Most often professional tendovaginites appear in construction workers - bricklayers, plasterers, painters, workers in the shoe industry, in electrical engineering - insulators, wrappers, etc.**

Test 8. **Constant microtrauma, overdistension of insufficiently vascularized tendon tissue and tendon sheaths under conditions of prolonged physical overvoltage lead to focal necrosis, ruptures, and destruction of individual fibrous bundles followed by hyalinization and calcification of the tendon. Reactive inflammation occurs in the nearby synovial tissues of the S-coli of the vagina. The transition of the inflammatory process to the articular bags with the formation of tendobursitis is possible.**

Test 9. **With professional tendovaginitis (tendinitis, tendosynovitis), patients complain of pains in the forearm and hands that occur during work with the hands. Pain sensations often limit movement in the wrist joint.**

Test 10. **With professional tendovaginitis (tendinitis, tendonitis-vitah), the swelling in the area of the affected tendons and their vagina is objectively determined. Palpation is determined by the pain points in the places of attachment of the tendons to the bone.**

Test 11. **In case of professional tendovaginitis (tendinitis, tendositicitis) radiographically, every third patient can find calcification points along the affected tendons, osteophytes in places of fixation of the tendon to the bone.**

Test 12. **Peripheral periarthrosis is a lesion of the skeletal-muscular structures involved in the movements of the shoulder sus-tava. This complex is composed of 4 articular joints, 15 muscles and 12 ligaments. The most common professionally caused humeroscapular periarthrosis is caused by a lesion of the tendons and muscles that form the rotating cuff of the upper arm — the hypochondral, supraspinatus, small round and subscapularis muscles. There is a degenerative and / or compression tendinitis of the supraspinatus muscle in combination with shoulder myofascial pain syndrome.**

Test 13. **Fractured periarthrosis occurs more often in men. Often it is preceded by trauma to the shoulder girdle. Characterized by complaints of pain in the back, in the shoulder in the region of the deltoid muscle.**

Test 14. **With humeroscapular periarthrosis it is possible to limit the rotation and removal of the shoulder between 60-1200. The initial lead is painless, further causes pain. Movement in the shoulder joint with active resistance to this movement causes the appearance or intensification of pain. The introduction of novocaine into the subacromial bag reduces pain, increases the volume of active movements in the joint. Passive movements in the shoulder joint are limited only slightly, by no more than 100.**

Test 15. **Occupational epicondylosis or shoulder tendonperiosteitis occurs when performing work with intense pronation and supination of the pre-shoulder, extension-flexion at the elbow joint. This disease is characteristic, but for construction workers - painters, bricklayers, bricklayers.**

Test 16. **In case of professional epicondylosis or tendonitis of the shoulder, prolonged overload leads to dystrophic changes in the tendons of the forefoot supinator, the extensors of the hand and fingers, and the gonchalusculus muscle in the place of their attachment to the lateral epicondy.**

Test 17. **During professional epicondylosis or shoulder tenodioperitis, complaints of constant aching pain in the elbow joint, aggravated by extension, supination of the forearm, are characteristic. Because of the pain, they cannot hold a brush that is unbent and clenched into a fist. Difficult to lift and hold heavy objects with an outstretched hand.**

Test 18. **In patients with professional epicondylosis or tendonteritis of the shoulder in patients, palpation determines pain points in the lateral or medial condyle of the shoulder. Lateral epicondylosis is manifested by severe pain during palpation of the shoulder. An X-ray examination reveals osteophytes, periosteal petrification at the site of attachment of the tendons to the condyles of the shoulder.**

Test 19. **Professional radiation styloidosis (de Kerven's disease) is a neurodystrophic pathological process that develops in the first canal under the dorsal ligament, in which the tendons of the short extensor and the long abductor of the thumb pass. Sickness can form in individuals who perform stereotypical ulnar leads of the hand in combination with thumb retraction. Most often, machine-workers, polishers, painters.**

Test 20. **Professionally-mediated ulnar styloidosis is characterized by the formation of a pathological neurodystrophic process in the sixth channel of the tendon retainer of the**

ulnar wrist extender, often in combination with compression of the external branch of the ulnar nerve. Occurs when performing stereotypical work associated with the maximum extension and abduction of the brush in the radial direction. The disease can be formed by workers of sewing, shoe manufacturing, installers.

Test 21. **A typical clinical manifestation of a professional radial styloidosis is pain in the circumference of the styloid process of the radial cosmetry, radiating to the forearm. A thickened, painful ligament is palpated over the styloid process. The painful sensations increase with an ulnar hand abduction, abduction and extension of the thumb. Patients complain about the impossibility of interconnecting the fingertips of the fifth and first fingers of the hand.**

*Test 22***. Radiographically, with ulnar styloidosis, osteo-phytosis is detected near the styloid process, compaction of the surrounding soft tissues.**

Test 23. **The clinical picture of professional ulnar styloidosis is characterized by pain associated with extension and abduction of the hand in the radial direction. Pain radiating to the IV and V fingers. Appears painful swelling, localized near the styloid process.**

*Test 24***. On radiographs in the initial period of a professional radial styloidosis, a thickening of the soft tissues surrounding the thyroid process is recorded. At later stages, osteophytes, cystic focal osteoporosis of the bone are detected.**

Test 25. **“Snap Finger” - stenosing tendovaginitis of the folds of the fingers - an occupational disease that occurs mainly in young people who perform manual work with long, significant pressure on the palmar surface of the hands. It can be formed by not-enough trained people, who started to work as an electric welder, puncher, cutter, polisher. Usually affects the fingers on one hand, more often I finger of the right hand, less often - several fingers of both hands.**

Test 26. **In the first phase of the disease there is pain in the palmar surface of the metacarpophalangeal joint, aggravated by the movement of the finger, pressing on this place. Then there are sensations of suddenly appearing obstacles to bending the finger. Soon, for the first time, a “latching” of a finger arises - fixation in a bent position, accompanied by pain. Fixation is eliminated by an arbitrary tension of the extensor muscles (active extension), after which the pain quickly disappears.**

Test 27. **In the second phase of the disease, "snapping" of the finger becomes frequent, accompanied by a pronounced pain reaction. It is not eliminated by arbitrary extensor contractions. To eliminate the "snap" the patient has to use the second hand (passive extension). After the release of the finger pain persists for a long time. Palpation of the pain point on the flexor tendon of the finger reveals a tight, painful uze-lok.**

Test 28. **In the third phase of the disease, "snapping" occurs very often. There are not only flexor, but also extensor fixation. They are eliminated with great difficulty. Sometimes, due to severe pain “snapping-in”, the disassembly fails, and the finger remains locked in a bent or unbent position. The pain points are located at the site of detection of a dense, diseased nodule on the flexor tendons and / or extensors of the affected finger.**
Radiographically, in the later stages of the disease, foci of consolidation on flexor tendons are detected near their fixation to the phalanges of the fingers.

Test 29. **Carpal or carpal tunnel syndrome is a chronic stenosing ligamentitis of the transverse carpal ligament. This is a fairly rare disease in occupational pathology. It can form during long, intensive work with fingers with constant tension of the muscles of the forearm.**

Test 30. **In this disease, due to overvoltage and constant traumatization, the wrinkling of the transverse ligament of the wrist occurs, forming the carpal tunnel together with the bones of the wrist and the palmar ligament. As a result of the narrowing of the carpal canal, there is a compression of the flexor tendons of the fingers and n passing through it. medianus.**

Test 31. **Complaints of numbness, paresthesia, pain in II – III fingers, aggravated by extension of the hand, are characteristic. Characterized by the forced flexion position of the hands, usually the right. Active movements of the kittens, especially the extension of the II – III fingers, cause sharp pains. The fingers are cold, cyanotic. The pain sensitivity of the skin on II - III fingers is reduced. With the percussion of the transverse ligament of the wrist, pain appears in the II-III fingers (Tennel's symptom).**

Test 32. **Only primary osteoarthritis can be professional. In accordance with the approved list, professionally determined is osteoarthritis with a lesion and dysfunction of the shoulder, elbow, and carpal joints. The etiological factor of occupational osteoarthritis is excessive mechanical (pressure) and functional overload of the joints, leading to the formation of a zone of subchondral sclerosis that affects the nutrition and regeneration of the cartilage of the articular surfaces, the formation of marginal osteophytes and bone growths in the form of nodes, synovitis and, ultimately, ankylosing of the affected joints. The joint may be jammed with a piece of detached cartilage.**

Test 33. **Formation of occupational osteoarthritis is possible in individuals working under conditions of physical overload when performing stereotypical movements aimed at seizing, lifting and moving weights with uneven distribution of loads on the joints of the hands. Often occurs in blacksmiths, builders, miners, porters, agricultural workers.**

Test 34. **For professional osteoarthritis is characterized by the absence of b-leu joints alone, at night. With the onset of physical activity, so-called "starting" pains arise due to reactive synovitis. A sudden, painful restriction of movement in the joint is possible as a result of a cartilage fragment (intra-articular "mouse") freely floating in the synovial fluid between the articular surfaces.**

Test 35. **Radiological signs of occupational osteoarthritis are subchondral osteosclerosis, flattening of articular surfaces, subluxation, marginal osteophytes, often in the form of ossified nodes. In severe cases of the disease, the cartilage lining of the articular surfaces may disappear with the subsequent formation of ankylosis of the joint.**

Test 36. **Professionally conditioned bursitis is a chronic aseptic inflammation of the synovial bags of the joints, which occurs as a result of prolonged overstrain and constant trauma of the joints. Often occurs when lifting and moving heavy objects with an emphasis on the knee, elbow, shoulder.**

Test 37. **Professional elbow bursitis can be formed by radio equipment installers, engravers, engravers. Knee bursitis preparatory - in parquet flooring, paving slabs. Felts are characteristic of molders, blacksmiths, loggers. Under-deltoid professional bursitis can occur in movers.**

Test 38. **During the formation of this disease as a result of the constant traumatization of the wall of the articular bags on the inner surface and in the cavity, numerous free bodies of cartilaginous density, calcifications appear. They can form dense tumors that limit the mobility of the joint.**

Test 39. **Bursitis usually does not cause restrictions on the mobility of the joints with the exception of the subdeltoid.**

Test 40. **Professional bursitis forms very slowly, and can take place only with long working experience. Gradually, a spherical tumor with a fluctuating content, dense, mobile, not welded to the skin, appears near the joint.**
For bursitis, the appearance of pain with a strong pressure on the oculostate swelling is typical.
An X-ray examination in the periarticular soft tissues revealed a rounded darkening, reaching several centimeters in the perimeter.

Test 41. **The coordinating neurosis is a functional disorder, manifested by a disorder of coordinated muscle movements that make it difficult to perform specific professional operations.**
It occurs in persons whose work is associated with the implementation of highly differentiated movements performed at an accelerated pace. This includes the professions of musicians, operators of electronic computers, draftsmen, accountants, office workers. The disease develops in highly skilled professionals engaged in intensive labor, usually against the background of general neuroticism.
The leading pathogenetic moments of this disease is the reconciliation of the functions of the central and peripheral nervous system, which can lead to the loss of a high degree of differentiation (writing, musical skills) acquired as a result of the professional activity of the motor stereotypical skill.

Test 42. **In the clinical picture of coordinating neurosis, the leading manifestation of the disease is the loss of the ability to perform complex professional motor skills. An attempt to perform them provokes the appearance of tremor, cramps, weakness, pain. At the same time there are no signs of organic damage to the central and peripheral nervous system. Any other motor acts that are not associated with subtle professional movements are performed easily and without any restrictions.**

Test 43. **Distinguish between shaking, sensitive, paretic and vessel-borne forms of coordinating neurosis.**

Test 44. **The trembling form of coordinating neurosis is characterized by the appearance of involuntary shaking of the hand during the execution of thin, differentiated movements of the hand, making it impossible to write, play the piano, type letters, etc.**

Test 45. **For the sensitive (neural) form of the focal neuro, the appearance of pain is typical when trying to perform coordinated, precise movements with the fingers. Often found in musicians - violinists, volunteers, guitarists.**

Test 46. **The paretic form of the coordinating neurosis is manifested by the extra-weak weakness, lethargy, uncontrollability of the fingers when trying to write a text - the pen "falls out" from the hands.**

Test 47. **The convulsive form of the coordinator nevrosa, which causes a violation of the ability to write, is most often encountered - “writing spasm”, playing musical instruments.**

EXPLANATION TEXTS
to test tests section V
“PROFESSIONAL DISEASES CAUSED BY EXPOSURE TO CHEMICAL SUBSTANCES "

Test 1. **For many toxic substances used in industry, antidotes have been developed that can neutralize the toxin in the body. There are** *etiotropic antidotes* **with the ability to specifically inactivate the poison that has entered the body and the toxic products of its metabolism. As well as** *pathogenetic antidotes*, **which have the ability to induce changes in the body of the victim, in their pathogenetic mechanism, opposite to the disorders caused by one or another poison.**

The group of antidotes *etiotropic* **actions include:**

• Donators of SH-groups - unithiol, sodium thiosulfate, mectides. These drugs are able to enter into low-toxic chemical compounds with thiol poisons - mercury, lead, arsenic, cyanides, which are then removed from the body through the urinary system.

• Complexes - thetacin-calcium, pentacin, D-penicylamine. Preparations of this group are capable of forming low-toxic soluble complex compounds of various metals - mercury, lead, copper, etc., with their subsequent elimination from the body with urine.

• Trimefatsin - binds and removes ions of rare-earth and heavy metals through the kidneys: beryllium, uranium, plutonium, zirconium, etc.

• Glucose (20% solution) - an etiotropic antidote for cyanide poisoning, methemoglobin-forming poisons.

• Desferal (deferoxyamine) - able to excrete iron ions from the body.

• Oxygen therapy (including hyperbaric) - used as an etiotropic agent for carbon monoxide poisoning, methemoglobin-forming poisons.

Test 2. **The group of antidotes of** *pathogenetic a***ction include:**

• Anticholinergic drugs - atropine, ganglioblokatory (pentamine, benzogeksony). Used in case of poisoning with phosphorus-organic compounds.

• Cholinesterase reactivators - dipyroxime, isonitrosine, alloxime. They are pathogenetic antidotes for intoxication with organophosphorus compounds.

• Methylene blue - a drug capable of maintaining anaerobic glycolysis under hypoxic conditions, including those caused by carboxyhemoglobinemia and carbon monoxide poisoning, methemoglobinemia caused by nitro compounds, and cyanide intoxication.

• Sodium nitrate is a drug that can cause the formation of methemoglobin, which is necessary for the inactivation of cyanides circulating in the blood (they are associated with ferric iron present in met-hemoglobin).

• Cytochrome C is used as a pathogenetic agent, allowing to a certain extent to compensate for severe hypoxia that occurs when carbon monoxide poisoning or methemoglobin forming nitro compounds.

• Enterosobenta - enterosgel, activated carbon, etc.

• Drugs of metabolic action - Essentiale, Lipostasil, multivitamins, Riboxin.

Test 3. **For acute professional intoxication, the following complex of urgent measures is carried out:**

• Removal of the injured person from the zone of exposure to the health hazard of the factor.

• Mechanical removal of toxic substances from the surface of the affected body: take off clothes, wash the body with water or with a solution of a neutralizing poison.

• Removal of ingested poison by repeated washing of the stomach.

• Neutralization of poisons in the digestive system through the introduction of poison binding substances.
• Neutralization of the toxins in the blood through the introduction of specific antidotes, as well as non-specific agents that are able to bind and excrete many types of toxic substances from the body, compete for receptor structures, preventing the fixation of toxic substances in organs and tissues.
• Formation of artificial diarrhea with salt laxatives with the aim of accelerated removal of poisons through the gastrointestinal tract and reduction of their reabsorption in the intestine.
• Nonspecific detoxification therapy by intravenous administration of physiological saline, 5% glucose solution simultaneously with the activation of diuresis by diuretic drugs with the aim of forcing the excretion of poisons through the urinary system.
• Carrying out inhalation of sodium bicarbonate solution with aerosols in order to normalize acid-base balance on the mucous bronchi.
• Introduction of glucocorticosteroids, antioxidants to protect organs and tissues from toxic damage.
• Conducting resuscitation measures according to the syndrome principle in case of shock, the presence of severe lesions of the respiratory organs, the cardiovascular system, disorders of oxygen transport, acid-base balance in the blood, the occurrence of acute renal failure.

Test 4. **Lead intoxication occurs in enterprises collecting lead batteries, persons serving lead batteries, installers of radio equipment using tin-lead solders, and workers in the production of crystal glassware (crystal is heavy glass containing lead compounds), pottery workers, where they can use enamels and glazes containing lead compounds.**

Test 5. **Lead and its compounds can enter the body through the respiratory tract, gastrointestinal tract, skin and mucous membranes. Under production conditions, lead intoxication most often occurs as a result of inhalation of dust or vapors of this poison, as well as through the skin when working with lead parts. Penetration of lead through the digestive system can occur when hygiene and safety regulations are not followed - eating at the workplace, insufficient cleaning of hands from lead contamination before eating.**
Lead and its compounds are a thiol poison that blocks the sulfhydryl groups of proteins.

Test 6. **Lead affects the blood, central, peripheral nervous system, parenchymal organs (liver, kidneys), mucous membranes, skin, skeletal bones.**

Test 7. **Lead compounds after penetration into the blood are able to be deposited in parenchymal organs (liver, kidneys, muscles), but to a greater extent in the bones as insoluble lead pyrophosphate. Under certain conditions, lead can leave the depot, as a result of which a picture of relapse of acute lead intoxication can occur even in the absence of external contact with this poison.**

Test 8. **Lead removal from the body occurs mainly through the urinary system, through the biliary tract and intestines. A small part of the lead circulating in the blood can be excreted by the sweat glands of the skin. During lactation, lead compounds can be excreted by the mammary glands.**

Test 9. **The severity of lead intoxication is caused not by the amount of lead deposited in various tissue structures, but by its concentration in the circulating blood.**

Test 10. **The most pronounced toxic effect of lead has on the bone marrow hematopoiesis system. Lead, blocking the thiol groups of proteins, causes deep disturbances in the system of metabolic processes responsible for the synthesis of porphyrins and heme. Lead, fixing itself in the erythrocyte membrane, reduces their life expectancy, contributing to their pre-transient hemolysis. There are direct, caused by the action of lead, or deep-mediated anemia-mediated dysfunction of the central and peripheral nervous system. The defeat by lead of the peripheral centers of the vegetative nervous regulation is accompanied by the formation of spastic-atonic changes in bowel function. This poison contributes to an increase in the blood content of sympathomimetic substances, which leads to an increase in blood pressure, vascular spasms, especially dangerous if they occur in the vessels of the brain.**

Test 11. **The clinical picture of chronic lead intoxication is derived from syndromes of blood damage, digestive organs, nervous system, skeleton.**
Chronic lead intoxication can occur in mild, moderate and severe forms.

Test 12. **Lead damage to the hematopoietic system leads to the formation of hypochromic, sideroachrestic, hypersidernemic, sideroblastic anemia. In the peripheral blood, the number of erythrocytes and the hemoglobin content in each erythrocyte decreases (hypochromic anemia). Due to the disruption of the hemoglobin synthesis process, iron is not absorbed (sideroahrestia) and its concentration in the blood increases (hypersidineremia). In the bone marrow, the number of cells containing deposited iron in the form of hemosiderin inclusions, sideroblasts, increases.**
One of the early signs of toxic damage to the hematopoietic system is a violation of porphyrin metabolism. For this reason, the content of protoporphyrin in erythrocytes increases, the excretion of mopping coproporphyrin and delta aminolevulinic acid increases. Excess porphyrin gives urine a characteristic reddish tint.

Test 13. **Criteria for the diagnosis of lead intoxication:**
• Professional route - work in conditions of exceeding the maximum allowable concentrations of lead aerosols in the air, other circumstances suggesting the occurrence of acute or chronic intoxication with this metal in the workplace.
• Asthenic syndrome, polyneuropathy, encephalopathy.
• Lead coloring - earthy-pale coloring of the skin due to anemia, spasm of skin vessels.
• Lead border on gums.
• Lead colic.
• Reticulocytosis, erythrocytes with basophilic granularity in peripheral blood.
• Hypochromic anemia.
• Increased serum iron content.
• Increasing the number of sideroblasts in the bone marrow.
• High content of protoporphyrin in red blood cells.
• Increased urinary concentrations of coproporphyrin and delta-aminolevulinic acid.
• Detection of lead in urine.

Test 14. **In connection with the increase in the blood porphyrins, vascular spasm, anemia, the skin of a patient with chronic lead intoxication acquires a characteristic grayish-earthy tint - lead coloring.**

Test 15. **In the peripheral blood of a patient with chronic lead toxicity, the number of reticulocytes, and young erythrocytes with residues of the nucleus in the form of basophilic granularity of the cytoplasm, increases. This testifies to the stimulation of**

erythroneogenesis processes due to the reduction in the life span of erythrocytes, their premature hemolysis in the spleen.

Test 16. **Lead damage to the digestive organs manifests quite specific symptoms.**
First of all, it is a constant, irritating feeling of the presence of a "hair" in the mouth, a metallic taste of its own saliva, nausea, and poor appetite.
In the oral cavity along the edge of the gums a "lead border" appears in the form of a blue-black strip along the edge of the gum. It is formed as a result of the reaction of soluble lead compounds released from saliva with hydrogen sulfide in the oral cavity. The resulting insoluble sulfur dioxide is deposited on the gums, painting them.
There are cramping pains in the abdomen around the navel, unstable stools with irregularly alternating periods of diarrhea and constipation. Patients may be disturbed by attacks of lead colic.

*Test 17***. In the case of attacks of lead colic, intense, cramping abdominal pains occur, spreading around the navel and in the epigastric region. In this case, the abdominal wall becomes sharply strained, retracted. Palpable individual loops of the intestine in the form of dense cords. The palpation of the abdomen reduces pain, as it facilitates the passage of content through the relaxed areas of the intestine.**

Test 18. **During lead colic, the feces become fragmented and take the form of sheep. Constipation may develop, not laxatives. Colic is caused by damage to the centers of the nervous regulation of intestinal motility. As a result, the individual intestinal loops appear in a state of paralytic relaxation, others in the spastic state.**
During lead colic, blood pressure increases, body temperature may rise, hemolysis of red blood cells may increase, and therefore the number of reticulocytes and erythrocytes with basophilic granularity in peripheral blood usually increases.

*Test 19***. Lead damage to the nervous system is expressed in the formation of asteno vegetative syndrome, polyneuropathy, encephalopathy.**
Astenovegetative syndrome is manifested by general weakness, fatigue, reduced emotional tone, memory impairment. The formation of a neurocirculatory triad is possible: hypothermia, bradycardia, arterial hypotension. In severe cases, psychopathological disorders may develop.

*Test 20***. Polyneuropathy in patients with chronic lead intoxication is a painless peripheral neuritis, weakness of the extensor muscles.**

Test 21. **Encephalopathy occurs in severe chronic lead intoxication. It is characterized by disorders of the cranial nerves in the form of anisocaria, twitching of individual muscle groups, ataxia, dysarthria. Convulsive attacks, cerebral vascular crises with hemiparesis, ophthalmoplegia may occur.**

*Test 22***. Lead skeletal damage is characterized by detecting the cartilage tissue of the metaphysis on the radiographs of the tubular bones (dense transverse stripes).**

Tests 23-25. **Lead intoxication can occur in mild, moderate and severe forms.**
In the mild form, asthenovegetative disorders predominate.
With moderate form of intoxication, a characteristic pale-earthy color of the skin, a lead border on the gums appears. There are moderately severe encephalopathy, a sensitive form of polyneuropathy, marked asthenovegetative disorders. Lead colic, hypochromic anemia is characteristic. Hemolysis of erythrocytes stimulates the appearance in the peripheral

blood of young forms — reticulocytes and erythrocytes with basophilic granularity of the cytoplasm.
The severe form is characterized by lead colic, severe lesions of the central and peripheral nervous system with sensory-motor disorders, the predominant weakness of the extensors of the hands and fingers, severe hypochromic, sideroachrestricheskoy, hyper-siderinemic, sideroblastic anemia.

Tests 26-28. **See test 13 (criteria for the diagnosis of lead intoxication).**

Test 29. **If it is necessary to remove lead ions from the body of the patient, antidote therapy is prescribed. Used complexion for oral administration:**
• Succimer - 10 mg / kg every 8 hours for 5 consecutive days. Then at 10 mg / kg after 12 hours for 14 days.
• D-penicylamine (cuprenyl) in capsules of 0.15 - 1 capsule 3 times a day in combination with taking multivitamin preparations.

Test 30. **If it is necessary to remove lead ions from the body of the patient, complexones are used for parenteral administration, which are used while maintaining renal excretory function:**
• Dimercaptol 3-5 mg / kg intramuscularly every 4 hours during the week in combination with intravenous injections of thetacin-calcium 150 mg / kg per day (for severe intoxication).
• Thetacin-calcium 100 mg / kg per day intravenously for 5 days (with mild and moderate forms of intoxication).
• Ethylenediaminetetraacetate (EDTA) disodium salt - 20 ml of 10% solution in 200 ml of 5% glucose solution 1 time per day for 3 days with a repeated cycle in 3-5 days.
• Pentacin 5 ml of 5% solution in 200 ml of 5% glucose solution 1 time per day for 3 days, repeating the cycle in 3-5 days.

Test 31. **In case of lead colic, antispasmodics (atropine), anesthetics (fentanyl), and antihistamines (chloropyramine, clemastine) are administered.**

Test 32. **If polyneuropathy and encephalopathy caused by lead intoxication occur, nootropil, vinpocetine, antioxidants (tocopherol, emoxipin) are prescribed.**

Test 33. **Tetraethyl lead (TPP) - an organic lead compound that is widely used as an additive to increase the octane number of low-grade gasoline - to produce so-called "leaded" gasoline. TPP easily evaporates even at zero temperature.**
In emergency situations acute poisoning of thermal power plants can occur. In connection with a pronounced cumulative (accumulative) effect, chronic poisoning of the power plant is possible with constant, prolonged contact with minimal concentrations of poison. The maximum permissible concentration (MPC) of the presence of TPP vapors in air is 0.005 mg / m3. Intoxication occurs by inhalation of TPP vapors, by ingestion by oral administration, as well as by ingestion of poison on the skin and mucous membranes.

Test 34. **TPP is a strong neurotropic toxin, which has the ability to easily penetrate the blood-brain barrier. The property of cytoplasmic and capillary venom, as well as the ability to increase serotonin content in the brain, leads to severe disorders of the central nervous system function. Pathological changes occur in the subcortical structures - in the reticular formation, the hypothalamus. Under the influence of TPP in the peripheral blood increases the content of acetylcholine.**

TES can circulate in the blood for several days unchanged. In the process of metabolic destruction, lead ions released from TPPs, circulating in the blood, can cause a disturbance of porphyrin metabolism, with the formation of sideroachrestic anemia (see lead poisoning).

Test 35. **In the clinical picture of acute intoxication of TPPs, there are three stages: initial, pre-culmination and culminating.**

Test 36. **The clinical picture of the initial stage of intoxication of the TPP is formed by a number of syndromes: asthenic, organic and preclirious.**

Test 37. **Asthenic syndrome is leading in mild intoxication of TPP. Its main symptoms are severe headache, marked general weakness, fatigue, insomnia, emotional instability. An increase in the content of acetylcholine in the blood is manifested by hypersalivation, bradycardia, and hypotension. Body temperature may drop. Arise-paresthesia.**

Test 38. **Organic or pseudoparalytic encephalopathic syndrome is characteristic of more severe acute TPP poisoning. The patient becomes euphoric, reminding his condition of acute intoxication with alcohols. In connection with the predominant lesion of the TES of the fronto-cerebellar part of the brain, the patient appears unstable gait, nystagmus, dysarthria, trembling fingers, twitching hands, legs, torso.**

Test 39. **When severe intoxication, a precaliorian syndrome forms, which is characterized by increased vegetative disturbances (hypersensitivity, hyperhidrosis), the appearance of sensations of the presence of hair in the mouth, and crawling of insects through the body. The patient begins to be disturbed by feelings of fear of persecution, imminent death. There are awesome hallucinations.**

Test 40. **Pre-Culminational Stage of Acute Poisoning of TPPs is characterized by aggravation of toxic brain damage, with the appearance of symptoms characteristic of organic damage to the central nervous system. Pronounced autonomic disorders are combined with general mental agitation, delusional state. On the background of a darkened consciousness appear frightening auditory, tactile, visual hallucinations. Patients become aggressive, dangerous to others.**

Test 41. **The culminating stage indicates the severity of acute poisoning of thermal power plants. Characterized by a sharp psychomotor agitation with darkening of consciousness, hallucinations, epileptiform convulsive attacks. In the absence of adequate assistance, the patient develops a comatose state with meningeal events, heart failure, and collapse, which are precursors of a lethal outcome.**

Test 42-44. **Chronic intoxication of TPP occurs with prolonged exposure to small doses of TPP, which has the ability to accumulate (accumulate) in the body. The most common cause of chronic occupational poisoning of thermal power plants is a violation of safety measures when working with leaded gasoline. The clinical picture of toxicosis develops very slowly.**
There are three stages of chronic intoxication TPP.
Stage I **These are the initial manifestations of the disease with neurosis-like symptoms. General weakness, fatigue, poor memory, insomnia begin to disturb the affected. The combination of a depressive state with increased irritability is characteristic. Gradually, patients begin to disturb nightmares, causeless fear. The senestopathic sensations of a foreign object ("hair") in the mouth, crawling of insects on the body appear.**

Stage II **Corresponds to the period of formation of toxic encephalopathy. The affected progressively decreases intelligence. Memory weakens. Becoming inadequate behavior. A variety of neurological disorders arise - trembling of the fingers, incoordination of movements (missed during the palmar test). Gait becomes unstable. Appear dysarthria, nystagmus. Increased tendon reflexes. Due to the accumulation of metallic lead in the body, sideroachrestrium anemia occurs.**
Stage III. **It is characterized by deep violations of the mental status with a predominance of psychomotor excitement and aggressiveness. At this stage, severe extrapyramidal (intentional tremor) and vegetative-sensory neuropathic disorders arise. Clinical manifestations of lead sideroahresticheskoy anemia are aggravated.**

Test 45, 46. **At acute intoxication of the TPP, caused by ingestion of the toxin, multiple gastric lavages with activated charcoal are performed. The wetted areas of the skin quickly washed with kerosene, and then thoroughly washed with soap and water to completely eliminate the smell of poisonous substances. To relieve psychomotor agitation, 10 ml of a 10% solution of hexenal, previously dissolved in 10 ml of water for injection, is injected. With the same purpose, it is possible to introduce 5-10 ml of a 5% solution of Barbamil. The elimination of convulsive syndrome contributes to the intramuscular injection of 5-10 ml of 25% solution of magnesium sulfate. For rapid removal from the body and metabolization of the circulating TES circulating in the blood, 400-800 ml of 5% glucose solution with ascorbic acid, saline, Ringer's solution are intravenously injected. Antioxidant therapy (ACE-vitamin complex).**
In chronic TPP poisoning, measures are taken to stop neurotic changes with the help of neuroleptics and tranquilizers. Detoxification and metabolic therapy is carried out. For this purpose, glucose solutions with vitamin C, cocarboxylase, piracetam are introduced. To stabilize cerebral hemodynamics, drugs from the group of calcium channel blockers (cinnarizine), cavinton, and pentoxifylline are prescribed. Acute and chronic intoxication of TPPs is a contraindication to the morphine, chloral hydrate, bromides.

Test 47. **In the chemical industry, aromatic hydrocarbons are the initial, intermediate or final products of organic synthesis. They are widely used in industrial environments as solvents for various chemical compounds. They are present as a quality pigment and / or solvent in the composition of paints, adhesives. Benzene and its homologues toluene, xylene are the most common; Nitro- and amidopro-benzene - aniline, ursol (p-phenylenediamine), nitrobenzene, trinitro-toluene. Pathogenic effects of aromatic hydrocarbons are most often exposed to workers in the chemical industry, as well as workers in the construction industry (painters).**
Intoxication with aromatic hydrocarbons are quite common, as these substances are widely used in chemical production. As organic solvents, they are used in the preparation of paints, adhesives. For this reason, malaria, installers, shoe factory workers, etc. are often exposed to the harmful effects of aromatic hydrocarbons.
Under production conditions, benzene in a vaporous form can enter the body through the respiratory tract. Possible penetration of benzene into the blood by diffusion through intact skin. In rare cases, oral poisoning with benzene and its homologs occurs. Most often it is the conscious use of benzene-containing liquids as an alcohol surrogate or for substance abuse. Benzene and its derivatives are able to accumulate in adipose tissue, in the bone marrow, in the cells of parenchymal organs, primarily in the liver.

Test 48. **Aromatic hydrocarbons undergo metabolic pre-formation of the liver into phenols, diphenols, which, after combining with sulfuric or glucuronic acid, become low-toxic compounds. Ben-ash and its homologues are partially unchanged, partially as metabolites excreted from the body through the lungs, biliary system, kidneys, sweat glands.**

Test 49. **Nitro and amide derivatives of benzene have a local carcinogenic effect and, when removed from the body through the urinary tract, can cause bladder cancer.**

Test 50. **Aromatic hydrocarbons - benzene and its homologues (toluene, xylene, styrene) are poisons of polytropic action. Able to affect the nervous system of the type of narcotic poisons.**

Test 51. **For chronic intoxication, a predominant lesion of bone marrow hematopoiesis with the development of hypoplastic states capable of transforming into tumor lesions of the bone marrow — hemoblastosis, hematosarcoma — is characteristic. Damage to the hematopoietic system can be direct, toxic, as well as mediated through a decrease in the body's content of vitamins B6, B12 and C. Benzene and its derivatives cause myocardiodystrophy and liver damage with the formation of fatty hepatosis during prolonged exposure.**

Test 52. **The clinical picture of intoxication with benzene and other aromatic hydrocarbons is formed from neurological syndromes and changes in the hematopoietic system.**
Asthenic syndrome is manifested by emotional lability, loss of ability for intensive mental labor, sleep disorders.

Test 53. **Polyneuritic syndrome during intoxication with aromatic hydrocarbons is characterized by autonomic disorders, sensitivity disorders. Vegetative disorders are manifested by a decrease in skin temperature, excessive sweating, swelling of the fingers, pulse lability, and blood pressure. Impaired sensitivity is especially pronounced in workers who come into contact with aromatic hydrocarbons with unprotected hands. Typical pain, paresthesia, reduction of skin sensitivity in a polyneuritic type.**

Test 54. **Typical signs of toxic encephalopathy syndrome when intoxicated with aromatic hydrocarbons include organic sym-ptomatics with dyscirculatory disorders, extrapyramidal hyperkinase. Psychotic abnormalities may occur.**

Test 55. **The syndrome of funicular myelosis is evidence of a toxic lesion of spinal cord structures. May occur as a result of vitamin B12 deficiency caused by benzene intoxication. Patients decrease deep muscular sensitivity, decrease Achilles reflexes. Coordination of movement is disturbed, weakness arises in the legs.**

Test 56. **With prolonged chronic intoxication with aromatic hydrocarbons, there is a consistent defeat of granulocytopoiesis, thrombocytopoiesis and erythropoiesis with the formation of aplastic anemia as a result.**

Test 57. **During acute and in the initial period of chronic intoxication, moderate leukocytosis can occur, which then goes into leukopenia.**
Trobocytopenia, indicating the severity of chronic benzene toxicosis accompanied by thrombocytopenic purpura, may be the cause of bleeding, including deadly hemorrhages in the internal organs, the brain.
Aplastic anemia at a certain stage of its development may have signs of hyperchromic megaloblastic anemia caused by a deficiency of vitamin B12.
Blast cells may appear in the peripheral blood, which is evidence of the transformation of chronic benzene toxicosis into leukemia. Most often formed acute non-lymphoblastic leukemia. Chronic leukemia may occur. Usually it is chronic myeloid leukemia, less often - chronic lymphocytic leukemia, true polycythemia.

Tests 58-60. **Acute intoxication with benzene can have three degrees of gravity:**
A mild degree of acute poisoning with aromatic hydrocarbons is like alcoholic intoxication.
With a moderate degree, signs of encephalopathy with loss of consciousness, muscular twitching, tonic and clonic convulsions are noted.
Severe intoxication is characterized by almost instantaneous loss of consciousness, respiratory arrest and death.

Tests 61-63. **Chronic intoxication with benzene and its derivatives has three degrees of severity.**
Mild chronic intoxication manifests itself as a neurasthenic or asthenic syndrome with autonomic dysfunction. It is characterized by leukopenia (up to 3.0-4.0 • 109 / l) with neutropenia, relative lymphocytosis, moderately severe thrombocytopenia (up to 150.0 • 109 / l).
The average severity of chronic benzene intoxication is manifested by polyneuritis, signs of funicular myelosis, encephalopathy. There is a pronounced leukopenia (2.0-3.0 • 109 / l), thrombocytopenia (less than 70.0-150.0 • 109 / l) with widespread hemorrhagic purpura, anemia caused by an erythropoietic germ in bone marrow and - staying focused.
Severe chronic intoxication is manifested by severe encephalopathy, deep leukopenia (2.0-0.7 • 109 / l) up to agranulocytosis, severe thrombocytopenia (10.0-50.0 • 109 / l) anemia, severe hemorrhagic Syndrome. In the sternal punctate, signs of devastation of the red bone marrow with its replacement by fat are panmielophthysis. Sometimes in the bone marrow and in the peripheral blood, bright cells can appear in large numbers - evidence of the occurrence of acute leukemia.

Test 64. **Criteria for the diagnosis of intoxication with aromatic carbohydrate-genera:**
• Professional route - proof of work in conditions of contact with aromatic hydrocarbons.
• Identification of signs of lesion of bone marrow hematopoiesis (signs of agranulocytosis, thrombocytopenia, aplastic anemia).
• The occurrence of acute "benzene" hemoblastosis or hematosarcoma.
• Signs of toxic encephalopathy, damage to the liver, heart.
• Exclusion using immunogenetic methods of leukopenia, various forms of thrombocytopenia, aplastic anemia and leukemia, not related to the professional activity of the patient.

Test 65. **Mercury is a liquid metal, hazard class I, easily evaporating at room temperature. Professional mercury poisoning is possible with dentists in the process of preparing amalgam for dental fillings, in the production of mirrors, fluorescent lighting lamps, and measuring instruments. Poisoning occurs when non-compliance with the rules of safety technology or malfunction of technological equipment.**
The maximum permissible concentration of mercury in the air of the working zone is 0.01 mg / m3 (maximum one-time), 0.005 mg / m3 is the mean shift.
Usually mercury enters the body through the respiratory and digestive organs. Inhalation of fumes or aerosols of metallic mercury and its compounds is most dangerous. The oral route of onset of intoxication is possible when soluble mercury compounds are dropped into the mouth. Metallic mercury is a little hazardous, since it is not absorbed in the digestive tract.

Test 66. **After inhalation of mercury vapor, it begins to circulate in blood, deposited in the liver, kidneys, and brain (pituitary, cerebellum). The majority of soluble chemical compounds of mercury, entering by the oral route, are very dangerous poisons. As a result of absorption in the intestine, they cause severe damage to the liver in the first place. Part**

of the mercury is deposited in the bones, liver, lungs. Deposition, exit from the depot and, then, re-deposit contribute to long-term circulation of mercury, chronic damage to internal organs, microvasculature of the vascular bed even after a single intake of poison in the body.

Test 67. **Metallic mercury after oral ingestion into the digestive tract is usually excreted unchanged along with feces. The mercury compounds in the blood circulating in the depot are gradually removed from the body through the biliary system and the intestines, as well as through the urinary tract, salivary, sweat glands. Mercury ions can be excreted in breast milk.**

Test 68. **Mercury is a classic thiol poison - it blocks the sulfhydryl SH-groups of proteins.**
Interacting with the sulfhydryl groups of proteins, mercury ions disrupt many vital metabolic processes. As a result, functional and then degenerative changes occur primarily in the central nervous system. Myelinated membranes of the nerve trunks are affected. There are various pathological changes in the parenchymal organs - the liver and kidneys. Blockade by mercury ions of renal carbonic anhydrase causes polyuria. In the last century, diuretic preparations containing mercury were widely used.

Test 69. **Acute mercury poisoning in production practically does not occur. They are manifested by neurotoxicosis in the form of intense headache, nausea, vomiting, severe weakness, adynamia. In the affected case, there is a metallic taste in the mouth, salivation, abdominal pain, and sometimes bloody diarrhea, polyuria. Ulcerative stomatitis and gingivitis are typical.**

Test 70. **Mercury erethism is accompanied by unusual behavioral reactions in the form of extremely pronounced timidity, embarrassment, strong emotional agitation with palpitations, reddening of the face, sweating, even in familiar surroundings, and among familiar people.**

Tests 71-73. **Chronic intoxication has been characteristic of individuals for many years in contact with production conditions with mercury and its compounds.**
Chronic mild mercury poisoning is characterized by the emergence of vascular dystonia with a neurosis-like syndrome in the form of pathological "embarrassment", emotional instability, vasomotor hyper-reactivity. Patients experience feelings of metallic taste in the mouth, memory decreases, physical disability falls, sleep is disturbed, and "causeless" attacks of severe headaches occur. Clinical manifestations of intoxication are possible in the form of mercury gingivitis with a purple rim on the gums, gastric dyspepsia, unstable blood pressure, tachycardia, small amplitude tremor of the fingertips.
Chronic moderate mercury intoxication occurs during prolonged contact with mercury aerosols, the concentration of which in the air is 3-4 times higher than the maximum permissible level. It is characterized by organic disorders in the central nervous system with pronounced manifestations of mercury erethism.
A complex asymmetric extrapyramidal, large-scale intentional tremor (flapping) appears against the background of small-amplitude asymmetric functional tremor. There is a sensitive form of polyneuropathy. The release of mercury with saliva causes gingivitis with an intense lilac rim on the gums, stomatitis. Clinical and biochemical signs of toxic hepatitis are recorded. According to the results of an objective, ECG, EchoCG studies, myocardial dystrophy is detected. In general, the analysis of blood reveals a tendency to eosinophilia.
Chronic severe mercury poisoning is manifested by toxic encephalopathy, persistent organic changes in the nervous system. Mercury erethism reaches extreme severity. Extrapyramidal large-expanding tremor becomes generalized.

Test 74. **Criteria for the diagnosis of mercury intoxication:**
• Professional route, certifying long-term contact in production with mercury and its compounds.
• Clinical manifestations of mercury eritism.
• The presence of complex mercury intentional tremor in combination with functional tremor.
• Detection of elevated levels of mercury in the blood and urine.

Test 75. **The following groups of drugs are used for the purpose of intensive elimination of mercury from the body:**
• Etiotropic therapy with unithiol - 5% solution of 5 ml intramuscularly 1 time per day for 5 days. After a break of 3-5 days with continued excretion of mercury in the urine, the treatment cycle is repeated. Instead of unithiol, succinimer can be used parenterally at 0.3-0.5 daily for 5 days.
• Disintoxication therapy with forcing diuresis: intravenous taline injection of hemodez, reopolyglucine, Ringer's solution, 5% glucose solution up to 500 ml per day in combination with 40 mg of furosemide for 3 days in a row.
• Elimination of dysmetabolic shifts: balanced complex multivitamin preparations, Essentiale-Forte, 2 capsules 3 times a day for 30 days.
• Means to improve the metabolism and blood supply of the brain: Aminalone, Piracetam, Stugerone, Cinnarizine.
• Restoration of adaptive adaptive reactivity: the minimum (adaptogenic) dosages of tinctures of ginseng, Chinese limonnika, zamaniha.
• Normalization of sleep and correction of behavioral disorders: Valocordin, Corvalol.
• Restorative treatment: hydrogen sulfide, pine and sea baths, ultraviolet radiation, physical therapy, psychotherapy.
• Spa treatment in Pyatigorsk, Sernovodsk, Matsesta.

Test 76. **Up to 40-50% of all agricultural pesticide poisonings associated with organophosphate pesticides.**
Common representatives of this group of substances are insecticides - dichlorvos, butifos, karbofos, metaphos, chlorophos.
Enters the body through the respiratory system, digestive tract, non-damaged skin. Metabolized in the liver. Partly in the native form, partly in the form of metabolites leaving the body through the kidneys, intestines
In the pathogenesis of organophosphate insecticide poisoning, the key point is the blockade of phosphodiesterase, an enzyme that destroys acetylcholine, the main mediator in muscarinic (M) and nicotine (H) cholinergic synapses of the central and peripheral nervous system.

Test 77. **The majority of insecticidal drugs used in household and in agriculture with an organophosphorus structure are not poisons of direct action. Only after metabolic transformations in the liver do these substances begin to possess the properties of the phosphodiesterase blocker, a phenome of “lethal” synthesis.**
As a result of the blockade of phosphodiesterase, a large amount of acetylcholine accumulates in synapses. There are M-cholinergic reactions in the form of constriction of the pupils, bronchospasm, bronchial hypersecretion, pathological activation of the secretory and motor function of the stomach.
In severe intoxication, H-cholinergic reactions also occur in the form of fibrillar muscular twitches, mental excitation.

Life-threatening central nervous systems, paralysis of striated muscles, pulmonary edema, acute myocardiodystrophy, and fatty liver necrosis are typical for severe poisoning.

Tests 78-82. **Organophosphate pesticide poisoning can be mild, moderate and severe.**
***In mild poisoning*, M-cholinergic reactions predominate. Those who suffer are worried about intense sweating, drooling, pain in life, nausea, vomiting with plentiful acidic liquid contents, expiratory dyspnea, cough with a large amount of light sputum. They become agitated and, at the same time, adynamic. Sharply narrowed pupils. Blood pressure rises. The pulse is increasing.**
Medium intoxication **is manifested not only by more severe M-cholinergic reactions, but also by peripheral and central H-cholinergic reactions. The skin of the affected become marbled pale. Fibrillation of the muscles of the tongue, eyelids, other muscle groups, involuntary jerky movements of the eyeballs occur. Difficult speech. Characterized by mental disorders with severe depression, hallucinations, twilight state. Possible fever up to 400C.**
Severe poisoning **proceeds in three stages.**
Stage of excitement with characteristic M-cholinergic reactions in the form of abundant sweat, drooling, tearing, bronchorea, difficulty breathing, blurred vision, abdominal pains on the background of mental arousal.
The convulsive stage is due to the involvement of peripheral and central H-cholinergic structures in the pathological process. Characterized by adi-namiya, twilight mental state. There are clonic-tonic muscle cramps. Severe asthmatic condition causes hypoxemia. Symptoms of toxic damage to the liver, kidneys, and heart appear.
Paralytic stage corresponds to the terminal period of severe intoxication. Affected are in a coma. Acidosis occurs. Breathing becomes wrong type of Cheyne-Stokes. Death occurs as a result of pulmonary edema, cardiovascular insufficiency, paralysis of the respiratory muscles.

Test 83. **Prolonged contact with organophosphate pesticides under production conditions can lead to chronic occupational toxicity. The risk of such damage in the first place is exposed to personnel storage of pesticides, agricultural workers engaged in transporting pesticides and processing plants.**
The disease begins with asthenovegetative disorders. General weakness, decrease in mental and physical working capacity, memory impairment, headache, dizziness appear and gradually progress. Diminished appetite. The injured lose weight. A red, persistent dermographism appears. Blood pressure decreases. Pulse frequency decreases. The liver increases.
On ECG, changes characteristic of myocardiodystrophy are recorded. The blood decreases the activity of the enzyme pseudo-cholinesterase.

Test 84. **With long-term chronic intoxication with organophosphorus pesticides, encephalopathy occurs. Sickly headaches, dreams with awesome nightmares begin to disturb patients. Sharply reduced intelligence. Visual and auditory hallucinations appear. Twitching of muscles, paresthesias appear and become permanent. In severe cases, spastic paralysis of various muscle groups are formed.**

Test 85. **In acute and chronic intoxication with organophosphate pesticides, the activity of pseudocholinesterase in the blood decreases.**

Test 86. **In substantiating the diagnosis of occupational toxicity with phosphorus organic pesticides, the following circumstances are taken into account:**
• Professional route confirming the likelihood of contact with the organophosphate pesticides under production conditions.

• The results of a hygienic survey of the victim's workplace, confirming the possibility of professional intoxication
• Characteristic clinic poisoning with organophosphate toxic chemicals.
• The study of the content in the blood of the affected pseudo-cholinesterase, the activity of which decreases with acute and chronic intoxication with organophosphorus pesticides in the blood.

Test 87. **M-cholinergic reactions in the form of pupillary constriction, bradycardia, pronounced activation of the secretory function (in acute poisoning) of the salivary, lacrimal glands, stomach, bronchorei. In the absence of cirrhosis of the liver or severe hepatitis with deep violations of the protein-forming function of the liver, only with organophosphate poisons in the blood of the victim, the activity of pseudo-coline este sharply decreases.**

Test 88. **In acute intoxication with organophosphate pesticides, emergency care is provided in the following volume:**
• Remove the injured person from the area exposed to pesticides.
• Remove contaminated clothing.
• Wash skin intently with soap and water, then treat with a weakly alkaline solution (2–4% sodium bicarbonate solution or 5–10% ammonia solution), and then 2–5% chloramine solution.
• Flush eyes with a stream of clean water and drop albumin into them (30% solution of sulfacyl-sodium).
• If the toxin is swallowed, clear the stomach with a probe. Give saline laxatives.
• Conduct antidote therapy.

Test 89. **In acute poisoning with organophosphate insecticides, the following antidote therapy is carried out:**
• Antidotes pathogenetic action:
- **M-cholinolytics:**

- Atropine sulfate 0.1% - 1-4 ml subcutaneously, in severe cases, intravenously. The drug is stopped to be injected if the patient's pupils are no longer constricted.
- **Central-acting cholinesterase reactivators:**

- Alloxime 0.075 - the contents of the ampoule are dissolved in 1 ml of water for injection and injected intramuscularly. This is the main anti-pill in the treatment of moderate and severe intoxication with phosphorus-organic compounds.
- **Cholinesterase reactivators others:**

- Isonitrosin 40% - 3 ml - intramuscularly or intravenously to 3 g per course of treatment.
- Dipyroxime 15% - 1 ml intramuscularly or intravenously to 1 g per course of treatment.
- **Ganglioblockers in the occurrence of the affected H-cholinergic reactions:**

- Benzogeksony 2.5% - 1 ml, dissolved in 20 ml of 5% glucose, enter 1-2 ml under the control of blood pressure (there may be a sharp decrease in blood pressure).
- Dissolve Pentamine 5% - 1 ml in 20 ml of 5% glucose, inject 1-2 ml each under the control of blood pressure.
• Detoxification therapy:
- **Hemodez - 400 ml intravenously.**
- **Reopoliglyukin - 400 ml intravenously.**
- **Plasma - 200-400 ml intravenously.**

• Antioxidant drugs (Essentiale-Forte, lipostabil, tocopherol).

Tests 90, 91. **Organochlorine pesticides include chlorindane, hexachlorobenzene, heptachlor, polychlorocamphene. Previously, it was the leading group of insecticides - chemical compounds used to fight insects. Currently, organochlorine pesticides are used in agriculture less and less because of their high resistance to natural decomposition and, hence, significant environmental damage caused to the natural environment.**
Organochlorine pesticides are poorly soluble in water, but they dissolve well in organic solvents and fats. They are able to penetrate into the human body through the respiratory organs, the digestive tract, the undamaged skin. Excreted through the urinary tract, intestines, sweat glands, mammary glands (in nursing mothers).
Organochlorine pesticides have a general toxic effect due to their lipotropy, the ability to penetrate the lipid layer of the cell membrane and suppress the enzymes of the respiratory cycle, primarily cytochrome oxidase. Some substances from this group are capable of blocking the thiol (SH-) groups of proteins.
The most severe pathological changes in case of poisoning with organochlorine toxic chemicals occur in the cellular structures of the central nervous system and liver. Changes in the central nervous system are in many respects similar to those that occur in encephalitis with a predominant lesion of the subcortical region.

Test 92. **After a short period of time after the penetration of an organochlorine pesticide into the body, severe weakness, nausea, vomiting, intense headache, and dizziness occur. There is a fever up to 39-400C. In severe poisoning, general inhibition quickly occurs, mental disorders, trembling, twitching of various muscle groups, and clonicotonic seizures. There is decompensated metabolic acidosis, manifested by frequent, noisy breathing. There is shortness of breath, a feeling of lack of air, compression of the chest. Asthmatic attacks are possible. Soon there are anuria, jaundice, indicating kidney and liver damage. In acute poisoning in the peripheral blood of neuropenia, increased ESR. In the urine protein appears.**

Test 93-97. **The clinical picture of chronic intoxication develops gradually, after prolonged work in adverse conditions with chlorine toxic chemicals. Asthenovegetative, poly-critical, cardiac and hepatic syndromes are gradually formed.**
Asteno vegetative syndrome. **General weakness appears and progresses, physical and, especially, mental disability is reduced. They start to disturb headaches, dizziness, sweating, emotional lability, palpitations, interruptions in heart rhythm.**
Polyneuritic syndrome. **There are pain along the nerve trunks, trembling, muscle twitching. Impaired skin sensitivity. Sometimes there are disorders of vision.**
Cardiac syndrome. **There is a tendency to lower blood pressure. The pulse is increasing. Supraventricular and ventricular premature beats appear. There may be blockade of the cardiac conduction system. According to ECG and EchoCG, signs of myocardial dystrophy are recorded.**
Hepatic syndrome. **Episodes of hypoglycemic states appear. Sclera, skin become icteric. Objectively revealed moderate hepatomegaly. The blood shows moderate hyperbilirubinemia, increased activity of AST, ALT, LDH. Ultrasound reveals signs of steatohepatitis - an increase in liver volume, a diffuse increase in echogenicity of the parenchyma, moderately pronounced disorders of portal hemodynamics.**

Test 98. **In justifying the diagnosis of occupational toxicity with organochlorine pesticides, the following circumstances are taken into account:**
• Professional route, confirming the likelihood of contact with the organochlorine pesticides in production conditions.
• The results of a hygienic survey of the workplace of the victim, confirming the possibility of professional intoxication with chlorine organic pesticides.

• A characteristic clinical picture of acute or chronic organochlorine toxic chemicals.

Test 99. **For acute toxicity with organochlorine toxic chemicals, emergency care is provided:**
• Remove injured person from the toxic area.
• Remove contaminated clothing.
• Wash skin intently with soap and water, then treat with 2% sodium bicarbonate solution or isotonic sodium chloride solution.
• If eye damage has occurred, rinse them with running water for 10-15 minutes and drip 2-3 drops of 30% sodium sulfacyl solution.
• If irritation of the mucous membranes of the respiratory tract occurs, drip a 2% solution of ephedrine or sofradex into the nose.
• If there are symptoms of irritation of the larynx, trachea, bronchi (cough), inhalations of bronchodilators (salbutamol, berotok), 2% solution of sodium bicarbonate with 0.5% solution of novocaine are shown. It is advisable to prescribe mast cell membrane stabilizers (ketotifen), antihistamine drugs, glucocorticosteroids for the prevention of acute toxic pulmonary edema. Necessary to establish inhalation of moistened oxygen.
• If the toxin is swallowed, clear the stomach with a probe.
• With psychomotor agitation, convulsive syndrome, intravenous injection of hexenal.
• In case of acidosis, inject 200-400 ml of 2% sodium bicarbonate solution intravenously.
• Parenterally administer glucose solutions, vitamins C, B1, cocarboxylase, pyridoxine, calcium gluconate. The daily volume of infusion therapy is 6-8 liters. Is forced diuresis.
Test 100. **In the treatment of chronic intoxication with organochlorine pesticides, symptomatic therapy is carried out to restore metabolic processes in the affected organs. Course detoxification therapy (5% glucose solution, saline solutions, reopolyglucine) is shown. Daily oral intake of balanced multivitamin preparations is necessary. Hepatoprotectors are prescribed - Essentiale-Forte, Liposta-Bil, Lipamid. The sanatorium treatment in sanatoria of general profile is shown.**

Test 101. **Organo-mercury pesticides, such as granozan, merkuran, merkurgeksan are highly effective fungicides designed for use in agricultural production for the treatment of flax seeds and other crops in order to protect them from pathogenic fungi. Since 2000, these substances have been banned for importation, production and use in the territory of the Republic of Belarus.**
Substances of this type are able to penetrate the human body through the lungs, gastrointestinal tract, and skin. They can gradually accumulate in the body. Excreted very slowly mainly through the kidneys, to a lesser extent through the intestines.
Organo-mercury toxic chemicals, as well as inorganic mercury compounds, belong to thiol poisons - substances that block sulfhydryl groups of various enzyme proteins. In addition, they have capillary-toxic, direct neurotoxic and cardiotoxic effects. May cause dysfunction of the gonads and ovaries in women.
In the process of metabolic degradation of organo-mercury pesticides, mercury ions are released, which can be deposited in bones, liver, kidneys, and brain tissues.

Tests 102, 103. **The first symptoms of acute poisoning may be a metallic taste, a burning sensation in the mouth. Soon, progressive general weakness, headache, dizziness, nausea, vomiting occur. Severe thirst, intense abdominal pain, diarrhea, often bloodless, begin to trouble. There is hypotension.**
For more severe intoxication signs of damage to the nervous system. It becomes an unstable gait. Hands begin to tremble. Difficult swallowing. Decreased hearing acuity. Vision is impaired. Blindness may occur. Often there is a sudden loss of consciousness with non-arbitrary urination and defecation.

In the blood of the affected leukocytosis is found, increased ESR. In the urine is detected mercury.

Tests 104-108. **Clinical manifestations of chronic intoxication with organo-mercury toxic chemicals occur after a relatively short period — several weeks from the start of work in adverse industrial conditions. The clinical picture of the lesion is formed by asthenic-vegetative, polyneuritic, diencephalic-hypothalamic, cardiac, and hepatic syndromes.**
Asteno vegetative syndrome. **Manifested by headache, dizziness, decreased physical and mental disability, impaired memory, increased emotional lability.**
Polyneuritic syndrome. **Increased tendon reflexes. There is a trembling of the fingers. It is possible the formation of encephalomyeliosis and Couloneuritis with focal and diffuse manifestations, epileptiform convulsive seizures.**
Diencephalic-hypothalamic syndrome. Characterized by a violation of thermoregulation, increased thirst, polyuria, insomnia, frightening nightmarish dreams with battle scenes. Psychotic crises may arise with feelings of gratuitous melancholy, fear.
Cardiac syndrome. **Arterial hypotension, bradycardia, supraventricular and / or ventricular premature beats, myocardial dysfunction with signs of circulatory failure occur.**
Hepatic syndrome. **Objectively revealed moderate hepatomegaly. In biochemical samples, a violation of liver protein-forming function, slight hyperbilirubinemia, increased AST, ALT, LDH, gamma-glutamyl transpeptidase activity are detected. Ultrasonography reveals signs of steatohepatitis - an increase in liver mass, a diffuse increase in echo genesity, a depletion of the vascular pattern of the parenchyma.**
It is also possible to identify signs of impaired renal function, manifested by proteinuria, cylindruria, leukocyturia, moderate hematuria. Traces of mercury are recorded in the urine.
In the chronic form of intoxication, *hypochromic or normochromic anemia, leukopenia, and increased ESR* **are observed.**

Test 109. **The diagnosis of occupational toxicity of organic mercury toxicants is based on the following circumstances:**
• Professional route confirming the likelihood of contact with organic mercury pesticides in the workplace.
• The results of a hygienic survey of the workplace of the victim, confirming the possibility of professional intoxication with mercury-organic pesticides.
• A characteristic clinical picture of acute or chronic organ mercury toxicity poisoning.
• Detection of mercury in the urine of the victim.

Test 110. **When providing emergency care for poisoning with mercury-organic pesticides, fungicides should:**
• Remove the victim from the area contaminated with pesticides.
• Remove contaminated clothing.
• Wash skin with warm water, alcohol-soda solution.
• If a chemical enters the digestive tract, flush the stomach with 2-3 liters of water, give activated charcoal or another sorbent.
• Conduct antidote therapy (see below).

Test 111. **Antidote therapy in the treatment of intoxication with mercury-containing fungicide pesticides includes:**
• Unithiol - is administered intramuscularly at the rate of 1 ml of 5% solution per 10 kg of the victim's weight 3-4 times in the first day, 2-3 times in the second day and 1-2 times daily for the next 3-7 days.

• Pentacin - intravenous drip of 5 ml of 5% solution of a drug diluted in 200 ml of 5% glucose.

Test 112. **In the treatment of chronic intoxication with mercury-containing fungicide pesticides, a course of antidote therapy is carried out with unitio- 1 ml of a 5% solution once a day for 7-10 days. Unithiol can be used in the form of inhalation. A solution containing 1 ml of a 5% solution of unithiol and 2 ml of distilled water is poured into the inhaler. Assign 15-20 minute inhalations 1 time per day for 7 days. Perhaps the use of succimera - 0.3 g of the drug is diluted in 6 ml of 4% sodium bicarbonate solution. Succimer is administered intravenously 1 time per day for 7-10 days. Assign oral administration of calcium gluconate, a balanced multivitamin preparations. Essentiale-forte, lipamide, lipostabil are used for stopping the hepatic syndrome. Various methods of physiotherapy are widely used: galvanic collar, hydrogen sulphide baths.**

Test 113. **In industrial production, substances having a toxic irritant effect on the respiratory organs are widely used. These include chlorine gas, hydrogen chloride, nitrogen oxides, ammonia, sulfur oxides, hydrogen sulfide.**
Chlorine (hazard class I) **is a gas used in the textile, chemical, pharmaceutical industry, for disinfection, in metallurgy, and in rubber production. Of the chlorine compounds, it is necessary to indicate hydrogen chloride (gas), sulfuric acid (liquid), chloropicrin (liquid), bleach (powder). During their decomposition, the active principle is chlorine and its compounds. MPC chlorine - 1 mg / m3. Chlorine enters the body through the respiratory tract. It has a strong cauterizing, irritating and reflex effect on the respiratory organs and the mucous membrane of the eyes.**
Nitrogen oxides (nitrous oxide, nitrogen dioxide, "laughing gas", **III hazard class, MPC in the working area air in terms of nitrogen dioxide - 5 mg / m3) are a mixture of various gases of irregular composition resulting from the evaporation of fuming nitric acid or its action on metals and organic matter. At the same time, depending on the temperature conditions and the volume of the room, nitrogen oxides of various oxidation states can be formed - nitrous oxide (N2O), nitric oxide (NO), nitrogen dioxide (NO2). Occupational poisoning can sometimes be observed under adverse sanitary and hygienic conditions of working with fuming nitric acid, its action on organic substances (coal, wood, paper, matter, etc.), during etching of metals and engraving on metal, upon receipt of acids (sulfuric, nitric , chromic) and artificial fertilizers, during blasting, during the burning of celluloid, during electric welding, etc.**
Ammonia (IV hazard class, MPC in the working area air - 20 mg / m3) **- a colorless gas somewhat lighter than air, has a suffocating odor and a sharp pungent taste. Ammonia is used in the production of nitric acid, ammonium nitrate and ammonium sulfate, liquid fertilizers, organic synthesis of urea, dyeing fabrics, making mirrors, as a refrigerant in the refrigeration industry, as well as for degassing.**
Sulfur oxides (sulfur dioxide, sulfur dioxide, sulfur dioxide, **hazard class IV, MPC in the working area air - 10 mg / m3) is a colorless gas with a strong suffocating odor. Sulfur dioxide can affect workers in the extraction of sulfur, the processing of sulfur oil, fuel combustion, production of sulfuric acid, bleaching, preservation of various materials.**
Hydrogen sulfide (II hazard class, MPC in the working area air - 10 mg / m3) **is a colorless gas with a characteristic odor of rotten eggs. Hydrogen sulfide is formed and released during the decay of organic matter, as a by-product in gas and coke plants. Under production conditions, the release of hydrogen sulphide is possible in the extraction and refining of oil, in chemical laboratories, in tanneries, in the production of viscose fiber (artificial silk), and matches. Acute poisoning with fatal outcomes was observed during the cleaning and repair of the sewer network, cesspools.**

In agriculture, a saturated ammonia water solution is commonly used as a fertilizer, the vapors of which can cause severe toxic-chemical lesions of the respiratory organs.

Test 114. **All gases that have an irritant effect have practically the same pathogenesis of toxic-chemical damage to the organs of the breath. They cause irritation and burning of the mucous membranes of the upper respiratory tract, eyes. Irritant gases, which have a good solubility in water, are retained in the upper respiratory tract and, therefore, in the first place cause tracheobronchitis.**

Test 115. **Relatively slightly water-soluble gaseous substances (nitrogen oxides) are able to penetrate to the alveoli, causing toxic alveolitis, pneumonitis, acute toxic and chemical pulmonary edema (according to the International Classification of Diseases Revision 10 - toxic adult respiratory distress syndrome) .**

Test 116. **The main forms of damage to the respiratory organs of a toxic-chemical etiology during acute intoxication include: acute tracheobron-hit, acute toxic-chemical pulmonary edema.**

Test 117. **The main forms of damage to the respiratory organs of a toxic-chemical etiology in chronic intoxication include: pulmonary edema, alveolitis, pneumonitis, chronic bronchitis, pneumosclerosis.**

Tests 118-124. **In the clinical picture of acute toxic-chemical lung injury, there are four periods:**
Primary reaction **In typical cases, a suffocating laryngospasm and bronchospasm are acutely affected by victims of inhalation of an irritant gas. The primary reaction is especially pronounced with inhalation of water-soluble gases (chlorine, hydrogen chloride, ammonia). It is less bright when entering into the respiratory tract relatively weakly water-soluble gases (oxides of nitrogen).**
*Hidden period***. It occurs immediately after the initial reaction and can last from 1 hour to 2 days. At this time, the victim recovers good health, he seems to be fully recovered. This is a period of imaginary well-being. It has a longer duration by inhalation of slightly soluble gases in water.**
*Period of extensive clinical reactions***. It begins suddenly, often no-chew with acute toxic-chemical pulmonary edema or acute toxic-chemical tracheobronchitis. The latter option is typical for the destruction of well-water-soluble gases.**
Acute toxic-chemical pulmonary edema can occur in two variants - blue or gray.
The blue variant is characterized by the development of acute hypoxia and hypercapnia. It is formed as a result of severe intraalveolar exudation combined with obstruction of the small bronchi. The affected person suffocate, with difficulty inhaling and exhaling, coughing with frothy sputum. In the lungs, over all the fields, against the background of weakened breathing, wet rales of different calories are heard.
The gray version differs from blue by the combination of acute hypoxia with hypocapnia. In such cases, pulmonary interstitial tissue predominates. This variant of acute toxic-chemical pulmonary edema is characterized by the occurrence of hypertension in the small arteries, leading to acute overload of the right heart, cardiovascular failure.
Acute toxic-chemical tracheobronchitis is the least life-threatening manifestation of a period of developed clinical reactions. It can manifest symptoms of pathological changes in the upper respiratory tract - toxic-chemical rhinitis, pharyngolaryngotracheitis. In many similar cases, acute endobronchitis occurs with a toxic-chemical lesion of the mucous membrane of the large bronchi.

The period of the outcomes. **With a favorable clinical course, the total duration of the clinical course of toxic-chemical damage to the respiratory organs is at least 2-3 weeks. After the relief of acute pulmonary edema, the symptoms of alveolitis and pneumonitis persist for a long time. Aseptic toxic-chemical damage to the alveoli, interstitial tissue, bronchus can be complicated by infectious-inflammatory changes, which is one of the reasons for the chronicity of the pathological process in the lungs.**

Test 125. **Long-term exposure of the respiratory organs to chemicals with irritating toxic properties may cause chronic damage to the respiratory tract and lungs. Primary aseptic inflammatory process, as a rule, is complicated by infectious-inflammatory. Chronic bronchitis is formed, often obstructive. Toxic-chemical damage to the lungs may be complicated by bronchial asthma. Chronic inflammatory process in the interstitial tissue, alveolar septa causes diffuse pneumosclerosis, pulmonary emphysema. In turn, these changes are the cause of the formation of chronic pulmonary heart, pulmonary heart disease.**

Test 126. **Diagnosis of toxic-chemical damage to the respiratory organs is carried out taking into account the following circumstances:**
• Professional route, showing the possibility of a chronic, or acute (emergency) toxic-chemical lesion of the respiratory system.
• The results of the hygienic assessment of the working conditions of the victim, with the identification of the gaseous substance causing the occupational disease.
• A characteristic clinical picture of acute or chronic toxic-chemical lesions of the bronchopulmonary system.
• Results of laboratory and instrumental diagnostics, giving an objective assessment of the severity of respiratory injury, the state of compensation of the pulmonary system.

Test 127. **In case of acute toxic-chemical damage to the respiratory organs, during the period of the primary reaction, the following measures are taken:**
• Remove injured person from danger area.
• At the scene of an incident, the mucous membranes of the mouth, nasopharynx, and eyes are immediately washed with distilled water.
• Give plenty of warm drink.
• Conduct inhalation of 1-2% sodium bicarbonate solution.
• Intramuscularly inject antihistamine and analgesic drugs. With severe pain in the chest, the introduction of narcotic analgesics (morphine) is indicated.
• For severe lesions, hydrocortisone is injected parenterally (25 mg intramuscularly after 6-8 hours).
• In order to reduce the permeability of blood vessels, calcium gluconate is given - by 0.5 inside 3 times a day.

Test 128. **During the latent period of toxic-chemical damage to the respiratory organs, the injured need to be monitored in a hospital setting. If a pulmonary edema is suspected, an ECG, chest X-ray, and complete blood count are performed urgently. Subcutaneous administration with prophylactic medication (0.5 ml of a 2% solution) and atropine (0.5 ml of a 0.1% solution) is possible.**

Test 129. **The following treatment is carried out at the developed clinical stage in the event of the occurrence of a toxic-chemical pulmonary edema:**
• Oxygen is given in the form of 48% air mixture, pre-saturated with ethyl alcohol vapor.
• Inhalation of antifoamsilan defoamer (2-3 ml of 10% alcohol solution) through a nasal catheter.

• Morphine - 1 ml of 1% solution or promedol - 1 ml of 2% solution intravenously slowly. To eliminate the vagotropic effects of morphine, 0.5 ml of a 0.1% solution of atropine is simultaneously injected subcutaneously.
• Benzogeksony - 1 ml of 2.5% solution or pentamine - 1 ml of 5% solution in 20 ml of physiological solution intravenously at 1 ml per minute under strict blood pressure control. They are used only for edema of the "gray" type of the lungs with normal or elevated blood pressure.
• Furosemide - 60-120 mg or more (up to 200-300 mg) intravenously slowly. At the severity of the alveolar phase of pulmonary edema of the "blue" type (medium and large bubble rales in the lungs), 10-20 mg of furosemide is administered.
• Isosorbide dinitrate inhalation if systolic blood pressure is not lower than 90 mm Hg.
• Inhalation of salbutamol or fenoterol (berotek).
• Methylprednisolone - 90-120 mg intravenously.
• When blood pressure drops, dof-min is injected intravenously (250 mg is diluted in 200 ml of reopolyglucin).
• After the relief of acute pulmonary edema in order to prevent pneumonia, antibacterial drugs are prescribed.

Test 130. **Symptomatic treatment is indicated for the detection of chronic intoxication with gaseous irritant substances. Shi-roko apply physiotherapy - inhalation of sodium bicarbonate solution. Assign the reception of a balanced multivitamin complexes. In the event of the onset and aggravation of secondary infectious-inflammatory processes in the bronchopulmonary system, antibiotics, expectorant preparations, calcium gluconate, small doses of veroshpiron are used. Chest massage, breathing exercises, sanatorium-and-spa treatment have a good therapeutic effect.**

Test 131. **Carbon monoxide or carbon monoxide is a colorless gas without taste, color and odor, highly toxic gaseous substance, class IV hazard resulting from incomplete combustion (oxidation) of carbon. It is a component of the exhaust gases of internal combustion engines.**
Carbon monoxide poisoning is possible in boiler rooms, when testing engines of engine rooms of diesel locomotives, in cabs of other vehicles. The increased content of carbon monoxide can be in the air of the working area of some workshops of the ceramic, brick, cement, construction industry, metallurgy, mechanical engineering.
The MPC in the air of the working zone is 20 mg / m3.
Carbon monoxide enters the human body exclusively through the respiratory tract and is excreted unchanged with exhaled air.

Test 132. **Carbon monoxide, which entered the blood through the alveolar membrane, competes with oxygen for hemoglobin. Moreover, carbon oxide has a greater ability than oxygen to combine with hemoglobin. That is why, carbon monoxide poisoning can occur at very low (0.07%), significantly lower than oxygen concentrations in ambient air. In addition, carbon monoxide forms a stronger, hemoglobin compound, called carboxyhemoglobin, compared to oxygen. This compound is unable to carry oxygen.**

Test 133. **When a large amount of carboxyhemoglobin accumulates in the blood, it becomes unable, under normal atmospheric pressure, to perform the function of transporting oxygen from the lungs to organs and tissues. There is a genetic hypoxia of the internal organs of a person, which can lead to a lethal outcome. In connection with the blockade of carbon monoxide iron by intracellular cytochrome oxidase, tissue hypoxia may also occur. Carbon monoxide quickly penetrates the blood-brain barrier. It acts on the central nervous system, both directly and through hypoxia.**

Test 134. **The decomposition of carboxyhemoglobin accelerates with increasing content of carbon dioxide (carbon dioxide) in the blood, as well as under the influence of the ultraviolet part of the spectrum of sunlight.**
In chronic carbon monoxide intoxication, a protective reaction may occur: the blood plasma increases the content of non-heme iron, which is capable of binding carbon monoxide, thereby reducing the formation of carboxy hemoglobin.

Tests 135-137. **When the blood contains no more than 20–30% hemoglobin combined with carbon monoxide (carboxyhemoglobin),** *mild acute intoxication* **occurs. Infected appear general weakness, shortness of breath, throbbing headache, dizziness, nausea, often vomiting. The pulse is taking place. Blood pressure is normal.**
The increase in the content of carboxyhemoglobin in the blood to 31-40% corresponds to carbon monoxide *intoxication of moderate severity.* **Affected over-braked, drowsy. Breathing is frequent, superficial. Vomiting is possible. Pulse sharply speeded up. Blood pressure is reduced. In connection with hypocapnia, arterial hypotension, excesses of short-term loss of consciousness are possible.**
If the concentration of carboxyhemoglobin in the blood exceeds 41%, *severe carbon monoxide poisoning occurs,* **leading to the formation of toxic, hypoxic coma. Characteristic tonic and clonic convulsions on the background of decerebration rigidity. Involuntary urination and defecation. Cyanosis of the limbs, general hyperhidrosis. The complexion is bright scarlet (carboxyhemoglobin gives this color). Breathing is intermittent, maybe Cheyne-Stokes. Pulse - 110-120 per minute, hypotension, a tendency to count. Body temperature rises to 39-400C (hypothermia is possible), leukocytosis, a decrease in ESR. Possible death from respiratory paralysis.**

Test 138. **In acute intoxication with carbon monoxide, toxic lung edema can form. Bright pink infiltrates, bullous necrotic changes in the dermis, caused by carbon monoxide intracellular iron-containing enzymes of the respiratory cycle, often appear on the skin.**

Test 139. **Acute carbon monoxide poisoning leaves behind itself mainly neurological disorders in the form of peripheral polyneuropathy, motor polyneuritis, toxic encephalopathy with severe psychasthenia, a tendency to psychosis.**

Test 140. **In the clinical picture of chronic intoxication with carbon monoxide, the leading role is played by the syndrome of neurological disorders. Dysfunctions of the central nervous system occur, manifested by psychasthenia, autonomic disorders, angiodystonia. Complaints of general weakness, rapid physical and, especially, mental fatigue, shortness of breath with moderate exertion, constant headache, tinnitus, dizziness, palpitations, and a tendency to high blood pressure are characteristic. In severe cases, diencephalic crises can occur, severe myocardial dystrophy can form, which can lead to decompensated circulatory failure.**
Chronic intoxication with carbon monoxide is characterized by moderate compensatory erythrocytosis, elevated hemoglobin, and hyper-derinemia. The content of protoporphyrin in erythrocytes increases, the excretion of coproporphyrin and delta-aminolevulinic acid with mocha increases. In case of a mild course of toxemia, after elimination of the causes of a chronic lesion with carbon monoxide, full recovery is possible. Chronic chronic intoxication can cause deep organic changes in the central and peripheral nervous system, the cardiac muscle, which is able to progress even after contact with carbon monoxide is stopped.

Tests 141, 142. **Correct diagnosis and differential diagnosis of carbon monoxide poisoning from other toxicosis help:**
• Reliable data on the presence in the air of the working area of an injured person with a high content of carbon monoxide.
• Clinical signs characteristic of carbon monoxide intoxication.
• Detection of the carboxyhemoglobin in the affected person by the spectrometric method.
• High blood levels of non-heme iron, delta-aminolevulinic acid and coproporphyrin in the urine during chronic toxicity toxicity to carbon monoxide.

Tests 143, 144. **In acute carbon monoxide poisoning, the following measures should be taken urgently:**
• The injured person is brought to fresh air, warmed.
• Provides excess blood saturation with oxygen. Oxygenotherapy is preferably carried out in a chamber for hyperbaric oxygenation, since at elevated pressure the solubility of molecular oxygen in plasma increases, and it can reach tissues without combining with hemoglobin.
• Drugs prescribed:
- Ferkoven - a ferrous chelate compound capable of binding carbon monoxide. 5 ml of the drug is administered intravenously at the same time with intensive oxygen therapy.
- Methylene blue — 20 ml of a 1% solution, administered with a 20% glucose solution, is injected intravenously. The drug supports anaerobic respiration in the tissues.
- Cocarboxylase - injected in 0.05-0.1 intramuscularly or intravenously drip in order to maintain aerobic glycolysis.
- Cytochrome C - 0.25% solution on 5% glucose. Enter from 6 to 25 ml per day. It is an aerobic respiratory stimulant.
- Ascorbic acid - 5-20 ml of 5% solution together with 500 ml of 5% glucose solution intravenously. It has antihypoxic properties.
- Barbamil - 3 ml of 10% solution together with 1 ml of 1% solution of dimedrol is injected intravenously with psychomotor agitation, convulsions.

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ПРОФЕССИОНАЛЬНЫЕ БОЛЕЗНИ: ТЕСТЫ С РАЗЪЯСНЕНИЯМИ

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